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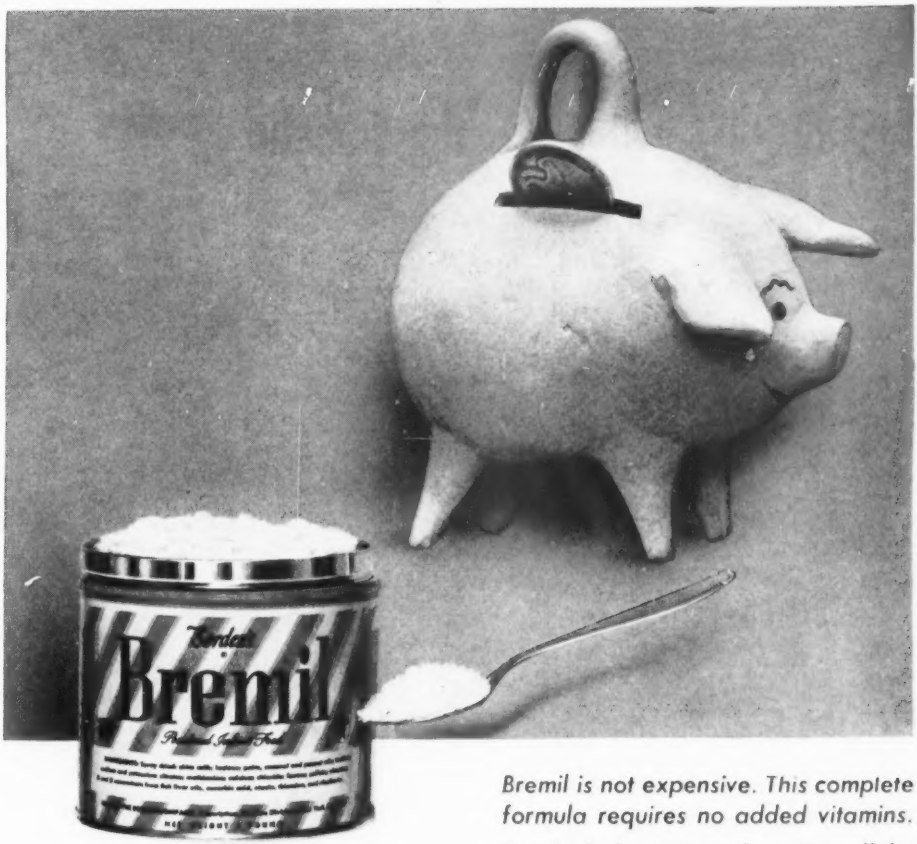

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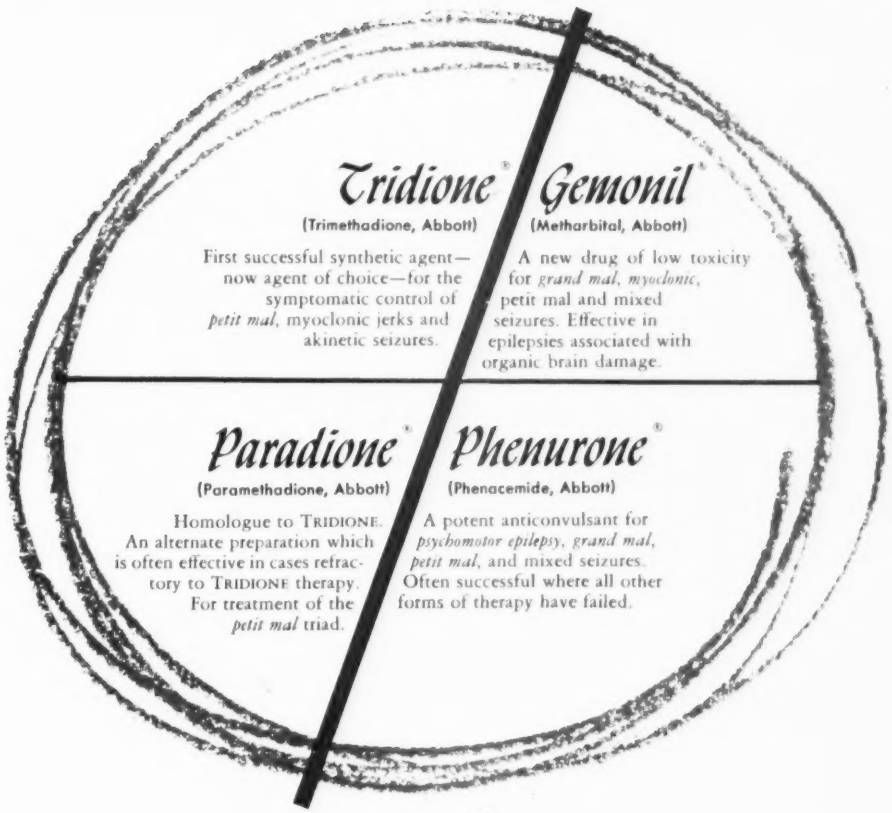
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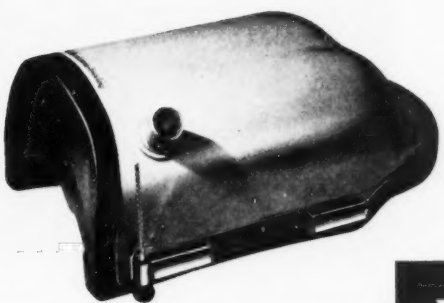

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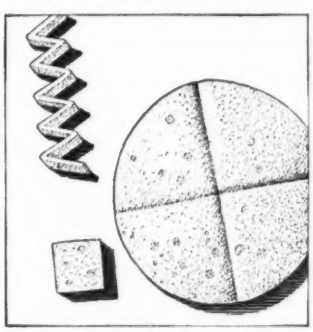

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GROWTH AND METABOLISM IN NORMAL AND THYROID-ABLATED INFANT RHESUS MONKEYS (MACACA MULATTA)

I. Growth and Metabolism in Normal Infant Rhesus Monkeys (Macaca Mulatta)

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LONG-TERM investigations designed to characterize the metabolic and endocrine features of growth and development in the human infant have been limited by a multiplicity of uncontrollable protean factors, included among which are heredity, a relatively slow rate of maturation, and our inability to prescribe the total environment of any child and his respective family. Use of primates other than man, while a recognizable escape from such limitations, has not been previously practicable due to the following considerations: (1) the need of a well-standardized primate colony on which sufficient growth and developmental data had been collected to warrant application of small numbers to such investigations; (2) the unavailability of techniques mandatory to successful management of member animals under experimental conditions; (3) the need for research tools, such as the application of tracer techniques and ultramicrochemical analyses, which were usually not within the practical domain of laboratories conducting such studies.

One of the authors (G. van W.) and her associates at Yale University have gathered growth and developmental data on more than 280 macaque infants born in her indoor colony during the last two decades. The basic features of her colony and the techniques employed have been incorporated in Farris' recent book, "The Care and Breeding of Laboratory Animals."[1]

Presented at the Twenty-Second Annual Meeting of the Society for Pediatric Research, May 7, 1952, at Old Point Comfort, Va.

From the Department of Pediatrics and Pediatric Section, Metabolic Unit of the University of California Medical School, (Dr. Pickering, Dr. Smyth, and Dr. Fisher); from the Department of Obstetrics and Gynecology, Yale University School of Medicine (Dr. van Wagenen).

This investigation was principally supported by a research grant from the Playtex Park Foundation and also received support from the Moore Estate, the Breon Research Fund of the University of California Medical School, and a U. S. Public Health Service grant to James Hudson Brown Memorial Fund of the Yale University School of Medicine.

1. van Wagenen, G., in Farris, E. J., Editor: Care and Breeding of Laboratory Animals, New York, John Wiley & Sons, Inc., 1950, Chap. 1.

As an initial application of these infants to pediatric research, we have chosen to investigate growth and metabolism in normal and in thyroid-ablated specimens during infancy and early childhood. The complete course of investigation is planned as follows:

I. Growth and Metabolism in Normal Infant Rhesus Monkeys (Macaca Mulatta).
II. Growth and Metabolism in Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta).
III. Growth and Metabolism Following l-Thyroxine Administration in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta).
IV. Radiosodium (Na^{24}) Space in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta).
V. Roentgenographic Features of Skeletal Development in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta).
VI. Iodine Metabolism in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta).
VII. Histopathology of Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta).

This paper presents recent studies of growth and development and correlated metabolic investigation in infant macaque monkeys. While physical, dental, and osseous features of macaque growth and development have previously been investigated,[2] there have been no reported studies relating associated metabolic features. Nitrogen metabolism has been studied in adult macaque monkeys under unrelated experimental conditions.[3] This laboratory has undertaken a study of growth and development in infant macaque monkeys during the first 140 days of life, correlating features of nitrogen, calcium, and phosphorus metabolism.

Calory, nitrogen, calcium, and phosphorus intakes were calculated throughout the period of study. In addition, 72-hour nitrogen-, calcium-, and phosphorus-balance studies were conducted at designated intervals for each of the animals.

Daily weighings, weekly general examinations, and physical measurements were made on each animal. Full body roentgenograms were taken at monthly intervals, and blood was drawn at appropriate intervals. Thus defined, normal growth and development will subsequently be compared to growth and development as modified by thyroidectomy in other infant macaque subjects.

METHODS

Care and Feeding.—All animals were individually housed indoors in small cages during early infancy and later in larger cages of galvanized wire mesh. For metabolic study, the animals were transferred to cages designed for separate collection of urine and stool specimens (Fig. 1).

Accurate intake measurement was accomplished through bottle feeding with weighed portions of special formulae. In addition, multivitamin drops and iron supplement were given in dosages comparable to those given newborn human infants.[4]

2. (*a*) Bast, T. H., and others: The Anatomy of the Rhesus Monkey (Macacca Mulatta), Hartman, C. G., and Straus, W. L., Jr., Editors, Baltimore, Williams & Wilkins Company, 1933. (*b*) Kennard, M. A.; Ruch, T. C., and Fulton, J. F.: The Housing, Care and Surgical Handling of Laboratory Primates, Yale J. Biol. & Med. **18**:443, 1946.

3. Wakeman, A. M., and Morrell, C. A.: Chemistry and Metabolism in Experimental Yellow Fever in Macacus Rhesus Monkeys, Nitrogen Metabolism, Arch. Int. Med. **46**:382, 1930. Rheinberger, M. B.: The Nitrogen Partition in the Urine of Various Primates, J. Biol. Chem. **115**:343, 1936.

4. Multivitamin and iron supplement: 0.3 cc. Zymadrops and 0.25 cc. Zymatinic drops were given every other day (The Upjohn Company, Kalamazoo, Mich.).

During the first month of life each animal was maintained solely on formula. Thereafter, each received protein milk during the night and banana milk during the day (see Table 1 for composition). Bottles were clamped to ring-stands outside the cage, with nipples entering the cage through a side mesh hole. These formulae provided more than 90% of calories ingested. Remaining calories were provided by supplement with apple, orange, or carrot. Caloric distribution was as follows: carbohydrate, 60 to 65%; protein, 12 to 15%, and fat, 20 to 25%.

Na^{24} Space.—Techniques for determination of Na^{24} space are those reported in detail in another communication.[5]

Roentgenographic Techniques.—These are reported in detail in a separate communication.[6]

Metabolic Methods.—Each balance study was conducted through a 72-hour period. Stool specimens were collected every 12 hours and preserved in 5% hydrochloric acid, while urine was

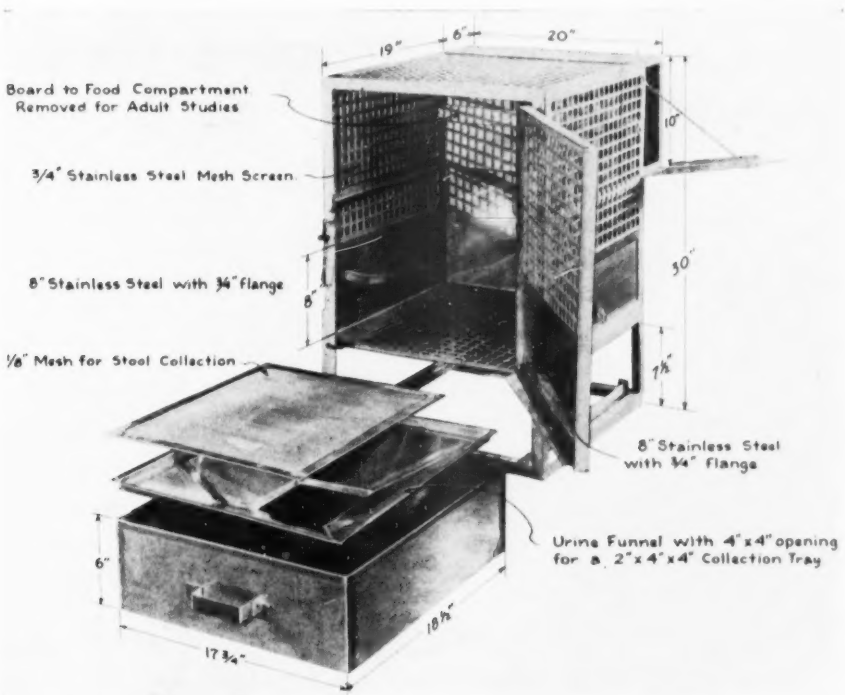

Fig. 1.—Metabolic study cage. The cage can be easily converted to suit adult macaque studies by removal of the partition to the posterior feeding compartment, the bottom of which has two 3-in. holes which house a sunken constant-filling water tray adapted from a similar water tray by Kennard, Ruch, and Fulton [2b] and a sunken food pan. Easy access to the food compartment is had through the side port shown. The stool-collection screen is covered with one layer of cheesecloth during study periods.

5. Pickering, D. E.; Scott, K. G., and Fisher, D. A.: Growth and Metabolism in Normal and Thyroid-Ablated Infant Rhesus Monkeys: IV. Radiosodium (Na^{24}) Space in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), A. M. A. Am. J. Dis. Child., to be published.

6. Lusted, L.; Pickering, D. E.; Fisher, D. A., and Smyth, F. S.: Growth and Metabolism in Normal and Thyroid-Ablated Infant Rhesus Monkeys: V. Roentgenographic Features of Skeletal Development in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), A. M. A. Am. J. Dis. Child., to be published.

allowed to collect over the entire 72-hour period in another preservative (thymol and acetic acid). All specimens were stored frozen. Techniques employed in urine and stool analyses were as follows: total urinary and stool nitrogen was determined by the method of Folin and Farmer [7]; urine and stool phosphorus, by the method of Fisk and Subbarow [8]; and urine and stool calcium, by the method of Michaels and co-workers,[9] except for one animal (C-794) for which urine and stool calcium was determined by a modification of the method of Sobel and Sobel.[10]

TABLE 1.—*Composition of Basic Diet* *

	Ingredients						Analysis					
Diet	Evap. Milk †	Karo	Water	Formula	Protein Supplement ‡	Banana Flakes §	Calories	CHO, Gm.	Fat, Gm.	Protein, Gm.	Ca, Mg.	P, Mg.
Formula	35	8	57	..	..	..	0.697	0.091	0.027	0.024	0.878	0.689
Protein milk	..	..	..	95	5	..	0.832	0.113	0.026	0.039	1.31	1.03
Banana milk	..	..	..	89	..	11	1.04	0.174	0.025	0.029	0.780	0.734

* Basic diet represents more than 90% of total calorie source.
† Courtesy of The Pet Milk Company, St. Louis.
‡ Meritene (courtesy of The Dietene Co., Minneapolis).
§ Kanana Banana Flakes (courtesy of the Kannenglesser & Co., New York).

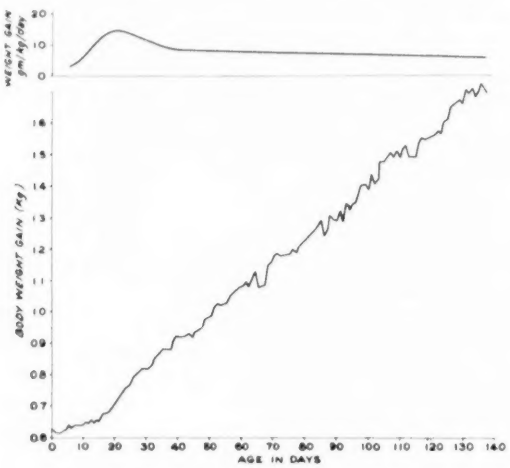

Fig. 2.—Weight graph of Macaque D-766 (male).

RESULTS AND COMMENT

Rates of body weight gain showed surprising similarity among animals during the period studied, as demonstrated by the weight graphs of four colony members presented below.

7. Folin, O., and Farmer, C. J.: A New Method for the Determination of Total Nitrogen in the Urine, J. Biol. Chem. **11**:493, 1912.

8. Fiske, C. H., and Subbarow, Y.: The Colorimetric Determination of Phosphorus, J. Biol. Chem. **66**:375, 1925.

9. Michaels, G. D.; Anderson, C. T.; Margen, S. S., and Kinsell, L. W.: A Method for the Colorimetric Determination of Calcium and Magnesium in Small Amounts of Urine, Stool, and Food, J. Biol. Chem. **180**:175, 1949.

10. Sobel, A. E., and Sobel, B. A.: Microestimation of Calcium in Serum, J. Biol. Chem. **129**:721, 1939.

Animal D-766.—This male animal was born in the Yale colony and subsequently flown to the University of California laboratory at the age of 40 days. As demonstrated in Figure 2, he had regained birth weight (630 gm.) at the age of 5 days and thereafter gained approximately 7.5 gm. per day. The rate of body weight gain (grams per kilogram per day) stabilized by 30 days (Fig. 2, top section), at which time he was gaining 10 gm. per kilogram per day. It declined thereafter at

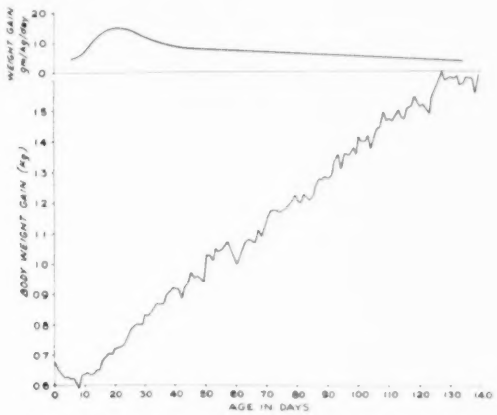

Fig. 3.—Weight graph of Macaque B-767 (female).

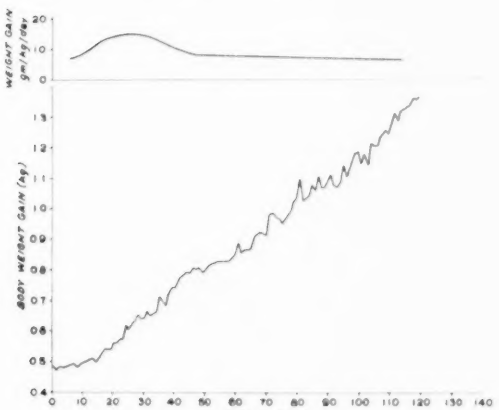

Fig. 4.—Weight graph of Macaque S-775 (female).

a nearly uniform rate until, at 140 days of age, he was gaining approximately at the rate of 5 gm. per kilogram per day.

Animal B-767.—This female animal was born at the Yale colony and subsequently flown to the University of California laboratory at the age of 39 days. Figure 3 demonstrates that she regained her birth weight (670 gm.) at the age of 14 days and thereafter gained approximately at the rate of 7.5 gm. per day. The rate of body weight gain (grams per kilogram per day) stabilized when she had reached 30 days of age (Fig. 3, top section), at which time she gained at the rate of 9 to 10

gm. per kilogram per day. Thereafter, weight declined at or near a uniform rate throughout the remaining period shown. She was gaining approximately at the rate of 4 gm. per kilogram per day during the last few days of study.

Animal S-775.—This female animal was born in the Yale colony and subsequently flown to the University of California laboratory at the age of 11 days. Figure 4 demonstrates that she had regained birth weight (490 gm.) at the age of 5 days and thereafter gained approximately 7.5 gm. per day. The rate of body weight gain (grams per kilogram per day) stabilized by 40 days, at which time she was gaining at or near a uniform rate of 8 to 10 gm. per kilogram per day, with a slight decline throughout the remaining period of study. Her smaller decline in rate of gain, as compared to that of the first two animals presented, was proportionate to her lower birth weight.

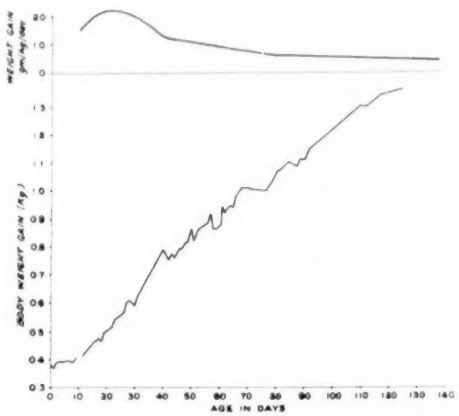

Fig. 5.—Weight graph of Macaque C-794 (male).

Animal C-794.—This male animal was born in the Yale colony and subsequently flown to the University of California laboratory at the age of 15 days. Figure 5 reveals that he regained birth weight (380 gm.) within four days after birth. The rate of body weight beginning at 30 to 40 days of age fell from approximately 10 gm. per kilogram per day to 5 to 6 gm. per kilogram per day at 140 days. As with the other animals, he gained approximately 7.5 gm. per day after resumption of birth weight.

All animals were born at term, and, though birth weights ranged from 380 to 670 gm., they fell within the normal range for this colony.[11] Attention is again called to the striking similarity of the rates of body weight gain shown by these four infants.

Characterization of the relative proportion of various organ masses to total body mass was sought through determination of the increment in the Na^{24} space.[12] The authors do not hold that, given a constant time for diffusion, a known quantity of

11. van Wagenen, G.: Personal communication to the authors.

12. Na^{24} space: the volume of distribution or fluid available to the isotope Na^{24} after its injection, expressed as per cent of body weight.

the radioactive isotope Na^{24} will singularly label extracellular water. The initially mentioned studies to be reported show that the time allowed for diffusion of Na^{24} bears a direct relationship to the relative size of the Na^{24} space.[5] A relatively larger Na^{24} space in very early infancy has similarly been described for the human infant.[13]

The Na^{24} space, determined 15 to 16 hours after injection of the isotope at any age after the initial 30 to 40 days of life, was found to be constant in all animals and to represent between 30 and 40% of body weight.

Anthropometric measurements—including crown-rump length, foot length, and head and chest circumference—followed a general pattern of growth similar to the tibial and femur increments which were determined roentgenographically with considerable more accuracy at 30-day intervals.

Femur length increased from 0.6 to 0.9 cm. in 30 days in the four animals studied, while over-all tibial length increased from 0.4 to 0.7 cm. Bone growth, so represented, was greater during the 90 to 120-day period than during the 60-to-90-day period. These facts are presented in greater detail along with many other roentgenographic features of skeletal maturation in a separate communication.[6]

Thus far, we have defined growth and development by an analysis of data relating body weight gain, increase of the Na^{24} compartment, and increment in linear bone growth. The following data are presented to correlate the above with intake and balance studies, showing the limits of nitrogen, calcium, and phosphorus retention at varying ages during the period of study. All data pertaining to this section are expressed in terms of a per kilogram per day basis, for example, calories per kilogram per day.

With constant exposure to protein formula during the night and banana formula during the day, previously described, voluntary caloric intake was subject to accurate analysis. It was observed to decline at a rate which was strikingly uniform and which closely paralleled the decline in rate of body weight gain after the animals' initial 30 to 40 days of life. Moreover, the total intake per kilogram per day did not vary among animals at any age.

The above points are presented in Figure 6. Also shown is the decline in caloric intake from 400 to 375 calories per kilogram per day common during the neonatal period to approximately 200 calories per kilogram per day at 140 days of age. It is again emphasized that more than 90% of all calories ingested were provided by standard formulae of constant composition (Table 1), which insured a fixed ratio between ingested calories, nitrogen, calcium, and phosphorus. Each calory ingested represented intake of 6 mg. nitrogen, 1.2 mg. calcium, and 1.0 mg. phosphorus. This relationship is also vividly shown in Figure 6.

Quantities of nitrogen, calcium, and phosphorus retained by these animals during such a uniform period of growth are next reported. It is emphasized that the only change required for conducting balance studies consisted in placing the animals in balance cages to which they had previously become accustomed. All other phases of care and feeding remained routine. Thus, the animals were not placed in a strange environment, nor fed a strange dietary by strange methods.

13. Perley, A. M.; Forbes, G. B., and Pennoyer, M. M.: Determination of Sodium ^{24} "Space" in Infants, Children, and Adults, J. Pediat. **38**:299, 1951.

Because of the qualitative and quantitative parallel shown by intake before and during the period of balance, data relative to excreta were felt to be more valid than would ordinarily have been the case.

Table 2 presents data on eight three-day periods of balance study conducted on three of the four animals.

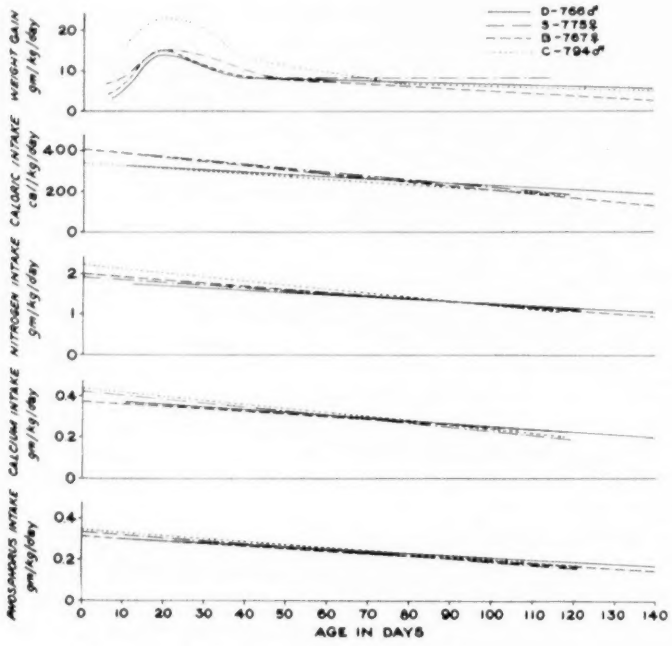

Fig. 6.—Daily intake graph.

TABLE 2.—*Balance Data on Infant Macaque Monkeys*

		Balance Data, Mg./Kg./Day								
		Nitrogen			Calcium			Phosphorus		
Animal	Age, Days	Intake	Output	Balance	Intake	Output	Balance	Intake	Output	Balance
D-766	96-99	1230	580	650	243	97	146	207	83	124
Male	131-134	1080	540	540	234	97	137	177	69	108
	136-139	1120	630	490	215	80	135	182	80	102
B-767	74-77	1200	564	636	252	84	168	209	85	124
Female	116-119	1390	825	565	272	91	181	227	85	142
C-794	58-61	1200	409	791	231	146	85	196	76	120
Male	73-76	1095	368	727	204	100	104	177	73	104
	77-80	1200	521	679	229	99	130	195	83	112

Relationships between nitrogen, calcium, and phosphorus retentions among animals are presented in Figure 7.

Based upon published data describing the chemical composition of human infants and children, various workers have attempted an indirect analysis of chemi-

cal growth through interpretation of balance data.[14] Because carcass analyses of macaque infants are not reported, and we are not free to assume the chemical composition of our animals at the outset of study, we must necessarily limit the discussion of chemical growth. Retentions of nitrogen at varying ages are shown in Table 3. Of the nitrogen ingested, between 10 and 15% was lost in the stool in all cases, and from 45 to 65% was retained, the remainder being excreted in the urine. Direct examination of the data reveals retention variation from 790 to 490 mg. per kilo-

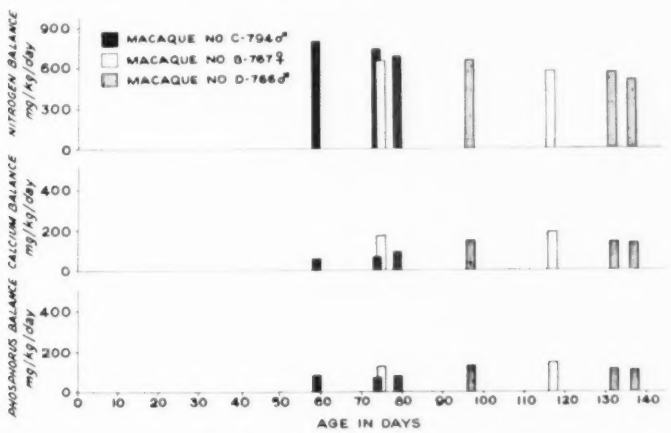

Fig. 7.—Metabolic balance data.

TABLE 3.—*Nitrogen Retained by Infant Macaque Monkeys*

Animal	Sex	Age, Days	Rate of Gain, Gm./ Kg. Day	Nitrogen Retained, Mg./Gm. Gain/Day
D-766	Male	95-100	6.5	100
		130-135	5.9	108
		135-140	5.9	98
B-767	Female	75-80	6	107
		115-120	4.75	105
C-794	Male	55-60	8	98.8
		70-75	7	103
		75-80	6.8	100

gram per day. When such retentions were adjusted to milligrams of nitrogen retained per gram of body weight gain per kilogram per day, variations were greatly diminished. The animals retained from 100 to 108 mg. nitrogen per gram of body weight gain per day.

Retentions of calcium and phosphorus shown in Table 4, similarly adjusted to milligrams retained per gram of body weight gained per day, are presented. A

14. Duckworth, J., and Warnock, G.: The Magnesium Requirements of Man in Relation to Calcium Requirements, with Observations on the Adequacy of Diets in Common Use, Nutrition Abstr. & Rev. **12**:167, 1942. Stearns, G.: The Mineral Metabolism of Normal Infants, Physiol. Rev. **19**:415, 1939.

nearly constant ratio existed in all cases between calcium and phosphorus retained. Most rapid rates of body weight gain were associated with a relatively smaller retention of calcium and phosphorus than those found associated with the slower rates of body weight gain. For example, Animal C-794 retained 10 mg. calcium per gram of weight gain at 55 to 60 days of age when he was gaining at the rate of 8 gm. per kilogram per day, while Animal B-767 retained 33 mg. calcium per gram of body weight gain at 115 to 120 days when gaining at the rate of 5.4 gm. per kilogram per day. The pattern of increased retention of calcium as per cent of such weight gain from the neonatal period through childhood has been described for humans.[14] Other factors known to influence calcium and phosphorus retention were made constant by uniform feeding, including dietary fat, calcium-to-phosphorus ratio in the diet, and vitamin D intake.

In conclusion, nitrogen retention paralleled growth closely and uniformly and represented approximately 10% of unit gain at any age between 40 and 140 days. While more variable, calcium and phosphorus retentions tended to increase, as per

TABLE 4.—*Calcium and Phosphorus Retained by Infant Macaque Monkeys*

Animal	Sex	Age, Days	Rate of Gain, Gm./Kg. Day	Retained, Mg. Gm. of Weight Gain	
				Calcium	Phosphorus
D-766	Male	95-100	6.5	22.5	19
		130-135	5.0	27.4	21
		135-140	5.0	27	20.4
B-767	Female	75-80	6	28	29.6
		115-120	5.4	33	26
C-794	Male	55-60	8	10.6	15
		70-75	7	16	16
		75-80	6.8	19.1	16

cent of unit gain, with increased age and corresponding decrease in rate of body weight gain.

Strikingly parallel relationships between rate of body weight gain and caloric intake have thus been defined for those macaque infants studied. During the period studied, growth seemed to be proportionately distributed. This was borne out especially by a constancy of Na^{24} space and nitrogen retained per unit gain.

SUMMARY

Investigations of various aspects of growth and metabolism were conducted on four infant macaque monkeys from birth through 140 days.

All animals gained approximately 7.5 gm. per day. Rates of gain stabilized by approximately 30 days at 10 ± 1 gm. per kilogram per day and thereafter declined at a uniform rate until 140 days, when weight gain was 5 ± 1 gm. per kilogram per day.

Voluntary caloric intake was uniform among animals and declined from 375 calories per kilogram per day in the neonatal period to 200 calories per kilogram per day at 140 days. Caloric distribution was constant throughout the study.

Balance studies revealed retention of 104 ± 4 mg. nitrogen per gram of weight gain per day. The ratio of calcium to phosphorus retained remained constant. Calcium retention averaged 25 mg. per gram weight gain after 75 days.

Miss Marjorie Cole and Miss Mary Gaylord gave technical assistance with this study.

GROWTH AND METABOLISM IN NORMAL AND THYROID-ABLATED INFANT RHESUS MONKEYS (MACACA MULATTA)

II. Growth and Metabolism in Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta)

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THIS COMMUNICATION presents a study of infant rhesus monkeys rendered athyroid by I^{131} administration. Its justification rests upon the premise that a better understanding of the effects of thyroidectomy in infant catarrhine primates might broaden our understanding of human cretinism. Aberrations in growth and development and correlated metabolic changes following thyroid ablation are reported. This report is one of a series on the growth and development of normal and thyroid-ablated infant rhesus monkeys.[1]

Previously reported studies on effects of thyroidectomy in the young rhesus monkeys are limited to those of Fleischmann and co-workers.[2] They observed that while no other physical effects were seen, one of the two rhesus animals which they surgically thyroidectomized at 8 months (the other of which died within a few days following surgery) showed no appreciably significant growth during a 21-week survival period.

From the Department of Pediatrics and Pediatric Section of the Metabolic Unit of the University of California Medical School.

This investigation was principally supported by a research grant from the Playtex Park Foundation and also received support from the Jefferson Macleod Moore Estate of the University of California Medical School.

1. (*a*) Pickering, D. E.; Smyth, F. S.; van Wagenen, G., and Fisher, D. A.: Growth and Metabolism in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta): I. Growth and Metabolism in Normal Infant Rhesus Monkeys (Macaca Mulatta). A. M. A. Am. J. Dis. Child., this issue, p. 1. (*b*) Pickering, D. E., and Fisher, D. A.: III. Growth and Metabolism Following *l*-Thyroxine Administration in Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), ibid., to be published. (*c*) Pickering, D. E.; Fisher, D. A., and Scott, K. G.: IV. Radiosodium (Na^{24}) Space in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), ibid., to be published. (*d*) Lusted, L.; Pickering, D. E.; Fisher, D. A., and Smyth, F. S.: V. Roentgenographic Features of Skeletal Development in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), ibid., to be published. (*e*) Pickering, D. E.; Fisher, D. A.; Scott, K. G.; van Wagenen, G., and Smyth, F. S.: VI. Iodine Metabolism in Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), ibid., to be published. (*f*) Crane, J.; Pickering, D. E., and Smyth, F. S.: VII. Histopathology of Normal and Thyroid-Ablated Infant Rhesus Monkeys (Macaca Mulatta), to be published.

2. Fleischmann, W.; Shumacker, H. B., Jr., and Straus, W. L., Jr.: Influence of Age on the Effect of Thyroidectomy in the Rhesus Monkey, Endocrinology **32**:238, 1943.

Earlier reports upon the effects of (surgical) thyroidectomy in adult rhesus monkeys are in considerable conflict. A number of authors have reported an absence of changes in physical appearance, appetite, activity, and behavior following thyroidectomy.[3] Among 10 animals studied by Jailer and co-workers,[3f] myxedematous changes were described in two spayed females. They reported that the animals increased in weight and coarse, dry, scaly skin, loss of hair, protuberant abdomen, and edematous lower eyelids developed. Similar changes in four thyroidectomized adult females, also spayed, had previously been reported by Burford and co-workers.[4] Though not specifying the species studied, Horsley, in the first reported studies of thyroidectomy in the monkey, aptly described myxedema.[5]

Previous reports specifically relating the metabolic effects of thyroidectomy have been limited to those by Fleischmann and co-workers [2] and Jailer and co-workers [3f] who reported that following administration of thyroxine in normal or in thyroidectomized animals there occurred a similarly precipitous drop in serum cholesterol and a creatinuria. Fleischmann and co-workers [2] were otherwise unable to detect any changes in serum cholesterol and urinary creatine excretion in adult animals following thyroidectomy. In contrast, Jailer and co-workers [3f] reported a transient hypercholesteremia following thyroidectomy in 6 of 10 animals which they studied. They coincidentally reported a 30% reduction in the circulation time of these animals.

While neither group reported a significant change in urinary creatine excretion following thyroidectomy, Jailer and co-workers [3f] were able, by retention studies, to demonstrate an increased capacity to retain exogenous creatine in such animals. Such creatine retention has been similarly described in the hypothyroid human subject.[6] While neither group reported changes following thyrotropic hormone administration in their thyroidectomized animals, both observed that its use in normal animals was followed by changes in serum cholesterol and urinary creatine excretion similar to those following thyroxine administration.[7] Fleischmann and co-workers [2] made the additional observation that the basal metabolic rate, when studied in two animals over a four-month period following thyroidectomy, remained unchanged at approximately 42 to 50 calories per kilogram of body weight per 24 hours, while in one thyroidectomized animal administration of 1 mg. thyroxine precipitated an increase from 44.1 to 56.8 calories per kilogram per 24 hours. Con-

3. (*a*) Footnote 2. (*b*) Vincent, S., and Jolly, W.: Some Observations Upon the Function of the Thyroid and Parathyroid Glands, J. Physiol. **32**:65, 1905. (*c*) Vincent, S., and Jolly, W.: Further Observations Upon the Functions of the Thyroid and Parathyroid Glands, ibid. **34**:295, 1906. (*d*) Carlson, A. J., and Woelfel, A.: On the Internal Secretion of the Thyroid Gland, Am. J. Physiol. **26**:32, 1910. (*e*) Halpenny, J., and Gunn, J. A.: Note on Extirpation of the Thyroid Gland in Monkeys, Quart. J. Exper. Physiol. **4**:237, 1911. (*f*) Jailer, J. W.; Sperry, W. M.; Engle, E. T., and Smelser, G. K.: Experimental Hypothyroidism in the Monkey, Endocrinology **35**:27, 1944.

4. Burford, T. H.; Allen, E., and Diddle, A. W.: Effects of Total Thyroidectomy Upon Experimentally Induced Menstruation in Mature Ovariectomized Monkeys, Endocrinology **20**:635, 1936.

5. Horsley, V.: A Recent Specimen of Artificial Myxœdema in a Monkey, Lancet **2**:827, 1884; Further Researches Into the Function of Thyroid Gland and Into Pathological State Produced by Removal of Same, Proc. Roy. Soc., London, s.B. **40**:6, 1886.

6. Thorn, G. W.: Creatine Studies in Thyroid Disorders, Endocrinology **20**:628, 1936.

7. Footnotes 2 and 3*f*.

trasting the negative findings which they reported in adult animals, these authors reported hypercholesteremia in the two young animals which they thyroidectomized at approximately eight months.[2]

With further reference to the metabolic effects of thyroidectomy in the rhesus monkey, it must be pointed out that despite Vincent and Jolly's negative report in 1906, they made the significant observation that sluggishness and anorexia occurred when their animals were taken from an artificially heated room and placed in a cold room. We have observed that temperature exerts a profound influence upon the manifestations of myxedema in the athyroid macaque monkey. Had Vincent and Jolly kept their animals in the colder room for more than a few days, they would undoubtedly have observed the development of classical myxedema.

In earlier reports thyroidectomy was uniformly accomplished by surgical means, and hypoparathyroidism usually followed.[8] Circumvention of that complication was attempted by Jailer and co-workers,[3f] when they reimplanted the animals' external parathyroid glands into the sternohyoid muscle. To avoid the hazards of surgery and to permit interpretation of data relative to the skeletal system and

TABLE 1.—*I^{131} Thyroid Ablation in Infant Macaque Monkeys*

Animal	Sex	Age	Wt., Gm.	Dose, Mc.	Element	Ablation
A-791	Female	10 hr.	535	1.0	I^{131}	Incomplete
		22 days	675	1.0	I^{131}	Complete
P-790	Female	10 hr.	473	1.0	I^{131}	Incomplete
		35 days	665	1.0	I^{131}	Complete
B-767	Female	58 days	1,040	0.10	At^{211}	Incomplete
		108 days	1,486	0.24	I^{131}	Incomplete
		162 days	1,890	1.50	I^{131}	Complete
S-775	Female	30 days	649	0.10	At^{211}	Incomplete
		80 days	1,040	0.32	I^{131}	Incomplete
		122 days	1,365	1.50	I^{131}	Complete

calcium and phosphorus metabolism, parathyroidectomy was not considered permissible in our infant macaque monkeys. We, therefore, elected to use I^{131} for thyroid ablation rather than risk surgical extirpation.

Growth and metabolic features following I^{131} thyroid ablation were investigated over a four-month period in two infants whose glands were ablated in the neonatal period. In addition, similar studies were conducted in two other female animals, one rendered athyroid at 4 months and the other at 5 months.

METHODS AND MATERIALS

All animals used in this and related reports were selected from Dr. van Wagenen's indoor colony of macaque monkeys at Yale University. Techniques of care and feeding and metabolic balance study have previously been reported in detail.[1a]

Thyroid Ablation.—Data relative to I^{131} ablation in the four animals studied are presented in Table 1. Radioiodine in isotonic carrier-free solution containing approximately 1 mc. per cubic centimeter was administered intraperitoneally in the doses indicated. The completeness of ablation was acertained by subsequent I^{131}-uptake studies, butyl extractable iodine determinations, and autopsy examination.

As in our previous study of normal infant macaque monkeys,[1a] caloric, nitrogen, calcium, and phosphorus intakes were calculated throughout the period of study. Blood was drawn at

8. Footnotes 2, 3, 4, and 5.

appropriate intervals for serum calcium and phosphorus analyses, determined by the methods of Sobel and Sobel,[9] and Lowry and Lopez.[10] In addition, 72-hour nitrogen-, calcium-, and phosphorus-balance studies were conducted at designated intervals for each of the animals, and urinary creatine and creatinine were determined on the same excreta by the method of Folin.[11] Techniques for nitrogen and phosphorus analysis in excreta have previously been reported.[1a] Techniques in calcium analysis of urine and stool specimens were those employed for serum analysis following pH adjustment of the ashed stool specimen.[1a] Daily weighings, weekly general examinations, and anthropometric measurements were made on each animal. In order to facilitate characterization of physical changes following thyroidectomy, photographs were taken at two-week intervals. Full body roentgenograms were taken at monthly intervals by techniques reported,[1d] and periodic determinations of the Na^{24} space were made.[1e]

RESULTS AND COMMENT

Data relative to thyroid ablation, per se, will be discussed before secondary growth and metabolic changes are presented.

Thyroid Ablation.—Each animal's thyroid was considered to have been ablated following the last dose of I^{131} given (Table 1). The reported observation of Hamilton and co-workers[12] demonstrated that At^{211} (Astatine, or Element 85) was concentrated by the thyroid in rats in sufficient quantity to ablate their glands, and ablation was originally attempted in these animals with that element (Table 1).

At^{211} was prepared by bombardment of bismuth with alpha particles on the 60-in. (152-cm.) cyclotron at the Crocker Laboratory. This radioactive element has a short half-life (7.5 hours), emits alpha particles rather than electrons and gamma rays, and each such nuclear particle arising from a nuclear disintegration dissipates an initial kinetic energy of 6 mev in about 50 μ of soft tissue,[13] while the beta rays emitted from I^{131} possess a maximum energy of 0.6 mev with a range of nearly 2,000 μ of tissue. The short half-life and associated high initial kinetic energy incident to the nuclear disintegration of At^{211} being dissipated in such a relatively small tissue mass might be interpreted as an advantage of this element over I^{131} for use in thyroid ablation.

Actually such features are a subtle hazard because they inadvertently result in a high radiation exposure of the hemopoietic system before thyroid concentration becomes significant. Administration of 100 μc of At^{211} precipitated a transient (10-day) but profound pancytopenia requiring antibiotic therapy in Animals S-775 and B-767. The 24-hour uptake of At^{211} by thyroid in these animals was approximately 30% in each case. Furthermore, in I^{131}-uptake studies conducted 30 days following that dose of At^{211} thyroid concentrations of I^{131} were within the range of normal for these animals (Fig. 1), demonstrating no appreciable thyroid effect by the At^{211} given and obviating the need for further radiation for complete

9. Sobel, A. E., and Sobel, B. A.: Microestimation of Calcium in Serum, J. Biol. Chem. **129**:721, 1939.

10. Lowry, O. H., and Lopez, J. A.: The Determination of Inorganic Phosphate in Presence of Labile Phosphate Esters, J. Biol. Chem. **162**:421, 1946.

11. Folin, O.: On the Determination of Creatinine and Creatine in Urine, J. Biol. Chem. **17**:469, 1914.

12. Hamilton, J. G.; Asling, C. W.; Garrison, W. M.; Scott, K. G., and Axelrod-Heller, D.: Destructive Action of Astatine 211 (Element 85) on the Thyroid Gland of the Rat, Proc. Soc. Exper. Biol. & Med. **73**:51, 1950.

13. Johnson, G. L.; Leininger, R. F., and Segre, E.: Chemical Properties of Astatine, J. Chem. Physics **17**:1, 1949.

ablation. Because of the complicating pancytopenia which followed the first dose of At^{211}, I^{131} was thereafter employed for thyroid ablation in Animals S-775 and B-767 and all other animals studied.

Ablation was again attempted at 80 and 108 days in Animals S-775 and B-767, employing 320 and 240 μc of I^{131}, respectively (detailed in Table 1). Gradation of dosage (320 μc per kilogram of body weight for S-775 and 120 μc per kilogram for B-767) was carried out in order that (*a*) secondary hemopoietic effects and (*b*) ablating efficiency might be determined following such dosage. Only minimal and unsustained (three to four days) myeloid suppression followed in either animal. During a 42-day period following the above dosage, some effects on the skeletal system were observed in animal S-775,[14] but no other clinical or metabolic effects were

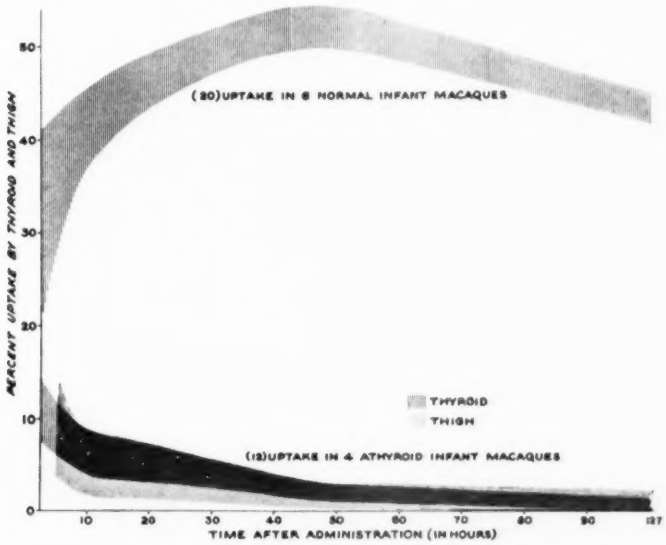

Fig. 1.—I^{131} uptake by thyroid in normal and athyroid macaque infants.

detected. Because this study centers around the effects of *complete* thyroid ablation on growth and metabolism, a final dose of 1.52 mc. of I^{131} was given to Animals S-775 and B-767 at 122 and 162 days, respectively. Three subsequent tracer studies conducted on each animal over 72-hour periods, employing 5 to 15 μc revealed I^{131} concentrations in the anatomical regions of the thyroid which never exceeded those in the region of the thigh (5 to 6%). A composite presentation of I^{131} uptake in thyroids of normal animals as compared to those observed in the experimental animals subsequent to complete ablation is presented in Figure 1.

It will be observed in Table 1 that Animals A-791 and P-790 received an initial dose of 1 mc. of I^{131} at 10 hours of age. That dose, based upon earlier observations on Animals S-775 and B-767 was considered to have been adequate for complete ablation. Though conducted before clinical evaluation of the initial attempt at ablation could be completed, I^{131} uptake studies at 20 and 34 days of age, respectively, revealed a low but significant uptake of approximately 10% by thyroid in

each animal. For that reason a second dose of 1 mc. of I^{131} was administered to each animal on the succeeding day. As with Animals S-775 and B-767, several uptake studies conducted on Animals A-791 and P-790 subsequent to their last 1 mc. dose of I^{131} revealed concentrations in the area of the thyroid comparable to those in the thigh areas. Such studies in these animals have also been included in Figure 1.

As detailed in other communications, serum-butyl-extractable iodine in the normal infant macaque monkeys studied has been found to range from 4 to 8 γ per 100 cc.[1e] When determined in Animals S-775 and B-767, 30 days following administration of At^{211}, the serum-butyl-extractable iodine was 4.5 γ per 100 cc. in each case, thus supporting the thesis that that agent in the dose given was inadequate for ablation. When determined 30 or more days subsequent to the final ablating dose of I^{131} (Table 1), in all four animals the serum-butyl-extractable iodine was 0 to 1 γ per 100 cc.

Complete autopsy examination of Animals S-775 and P-790, as well as confirming the presence of myxedema, revealed no gross or histological evidence of thyroid tissue and also revealed an enlarged anterior pituitary with a preponderance of large vacuolated and refractile pituitary beta cells.[1f]

TABLE 2.—*Serum Calcium and Inorganic Phosphorus in Athyroid Infant Macaque Monkeys*

Thyroid Status	Determinations	Calcium, Mg./100 Cc.		Phosphorus, Mg./100 Cc.	
		Range	Average	Range	Average
Athyroid	16	8.2-11.5	9.6	2.53-5.54	3.88
Euthyroid	17	8.1-11.4	9.4	3.08-5.62	4.27

The above series of findings were considered to represent conclusive evidence to support the presumption that I^{131} was capable of completely ablating the thyroids of infant rhesus monkeys in the doses given.

As a means wherein parathyroid integrity could be confirmed, periodic calcium and inorganic phosphorus analyses were carried out on sera from these animals over a four-month period following thyroid ablation. Tetany had not been observed in any case, and urinary excretion of calcium and phosphorus was not suggestive of hypoparathyroidism. Data obtained by analysis of 16 such serum specimens in the four athyroid animals were compared to data obtained by analysis of 17 similar specimens obtained from four normal infant animals of similar age and over an equal period (Table 2).

In the athyroid animals serum calcium averaged 9.6 mg. per 100 cc., while an average of 9.4 mg. per 100 cc. was observed in the normal group. Serum inorganic phosphorus values in the athyroid group similarly were not unlike those in the normal animals and again failed to suggest hypoparathyroidism, that is to say, there was no elevation of serum inorganic phosphorus in the athyroid group. Final conclusive proof was obtained following elective autopsy of Animals S-775 and S-790 when viable parathyroid glands were identified histologically in each case.

In summary, I^{131} in the doses given effectively ablated the thyroids of these animals without coincidentally rendering them hypoparathyroid.

Growth and Metabolic Changes Following Thyroid Ablation.—Behavior and general somatic features in each animal underwent striking changes following

thyroid ablation and resulted in development of a clinical picture not unlike that seen in the athyroid human infant. Making their appearance between 15 and 20 days following thyroid ablation, such changes were routinely pronounced after an additional 20 days and were most extreme in the younger infants. Following the advent of macroglossia and generalized myxedema (among the last signs to appear), labored breathing was not uncommon. When any of the athyroid animals were taken from the usually warm (22 to 24 C.) colony room and placed in a cooler room (18 to 20 C.) body temperature frequently dropped to below 35 C. (95 F.) within a few hours. In conjunction, their usually sluggish motor activity slowed further, and somnolence increased. Generally, lassitude and somnolence and apparent autism replaced alert inquisitiveness, agile mobility, and the gregarious nature common to normal infant macaque monkeys in this laboratory. As with

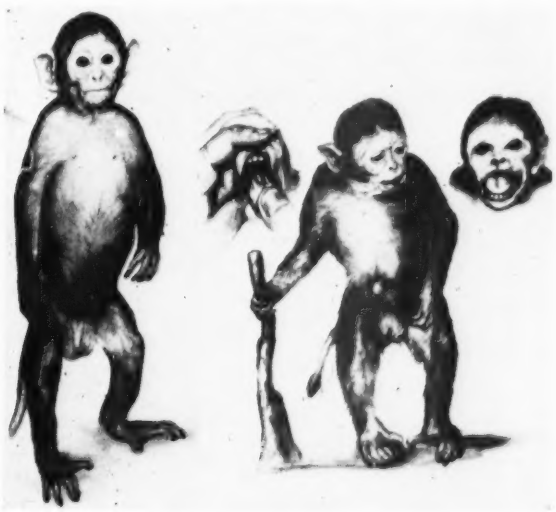

Fig. 2.—Left, normal macaque infant at 130 days of age. Right, athyroid macaque infant at 130 days of age with head views showing macroglossia and myxedematous paletoglossal and paletopharyngeal arches.

features described above, physical characteristics in the thyroid-ablated infant were slightly less prominent in the older group. In Figure 2 physical features of one of the two younger athyroid animals, A-791, are compared to those of a typically normal infant, C-794, by a composite illustration made from photographs and artist's sketches of the animals when they were approximately 130 days of age (Fig. 2).

A marked deficit in over-all growth was prominent following thyroid ablation. The animals usually assumed a seated or crouched position, with semiflexion of the lumbodorsal and cervical spine as well as the hips, elbows, and knees. The skin and subcutaneous structures became generally thickened (myxedema), and folds of skin were apparent in the face and low cervical regions. Hair was coarse, irregular, and without sheen and did not follow body contour. There was loss of hair on

the relatively shortened tail and, in the older animals, over the forearms as well. Delayed eruption of deciduous teeth was marked, as shown by the presence of only eight incisors in this athyroid animal at 130 days, six of which were present at 20 days. In the normal animals studied, all eight incisors had erupted by 35 days and between 16 and the full complement of 20 deciduous teeth usually were present at 130 days. Macroglossia and an associated enlargement of the palatoglossal and palatopharyngeal arches were pronounced. Such changes in these animals, along with myxedematous vocal cords, resulted in considerable compromise of the airway. There was development of an umbilical hernia, and the abdomen became doughy and protuberant.

It is of interest that the volume of body fluid available for distribution of Na^{24} in the myxedematous animals was equal to that in the normal animals,[1c] even

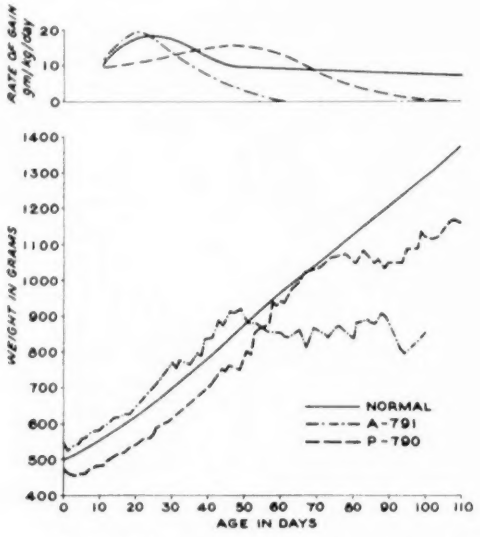

Fig. 3.—Body-weight graph of athyroid macaque infants.

though the former commonly experienced an appreciable weight loss within a few days following administration of *l*-thyroxine.[1b]

Skeletal effects of thyroid ablation consisted of a generalized and profound delay in linear bone growth and skeletal maturation, sellar enlargement, and a characteristic flattening and thickening of the basiocciput.[1d]

Thyroid ablation in these infants was followed within 30 to 40 days by almost complete cessation of body weight gain. In Figure 3 comparison is made between the body weight gain of Animals A-791 and P-790 during their first 110 days of life and that reported from this laboratory for normal infants over a similar period.[1a]

As noted in Table 1, Animals A-791 and P-790 had received an intial incompletely effective 1 mc. dose of I^{131} at 10 hours of age, and each was later completely ablated by a second 1 mc. dose at 22 and 35 days, respectively. Weight gain by Animal A-791 during the first 30 to 35 days of life was not unlike that reported

in our normal infants.[18] Thereafter, there was a rapid decline in rate of gain until her weight plateaued (at approximately 850 gm.) at 50 days of age (Fig. 3). The plateau in weight coincided with the appearance of pronounced myxedema. In Animal P-790 there was a delayed appearance of the marked acceleration in rate of gain which normally occurs within a few days following birth.[18] Such a delay was no doubt the result of a transient effectiveness of the initial dose of I^{131}. She, too, however, plateaued in body weight (1,050 to 1,100 gm.) 35 to 40 days after her second dose of I^{131}. Again, though actually appearing 10 to 15 days earlier in each case, myxedema became prominent when weight leveled off. Subsequent weight variation in either animal reflected variation in the degree of myxedema.

Body weight gain similarly ceased in Animals B-767 and S-775 approximately 35 days after I^{131} thyroid ablation at 162 and 122 days, respectively, again, when myxedema became prominent. Except for minor day-by-day fluctuation, body weight thereafter remained stable during an additional 100 days of study. Data describing the above may be seen in Table 3.

TABLE 3.—*Body Weight Changes Following I^{131} Thyroid Ablation (Animals B-767 and S-775)*

Animal	Age, Days	Weight, Gm.	Weight Gain, Gm.	
			Actual	Expected
B-767	162*	1,890	...	.....
	192†	2,140	250	200 ± 50
	222	2,350	...	200 ± 50
	252	2,150	...	180 ± 40
	282	2,200	50	160 ± 40
S-775	122*	1,370	...	.....
	152†	1,560	190	200 ± 50
	182	1,550	−10	200 ± 50
	212	1,530	−20	200 ± 50
	242	1,530	...	180 ± 50

* I^{131} thyroid ablation was complete at this age. Until this age, the animals were clinically euthyroid.
† Signs of athyrosis were prominent at and after this age.

Analysis of ingesta in each animal during the period of study revealed a voluntary restriction in caloric intake which was closely associated with the drop and plateau in body weight gain, restricted physical activity, and development of myxedema, all of which appeared 30 to 35 days following thyroid ablation. Figure 4 presents a comparison between the average rate of gain, caloric nitrogen, calcium, and phosphorus intakes in normal animals previously reported,[18] and similar data on athyroid Animals A-791 and P-790.

Following thyroid ablation at 22 days in Animal A-791, voluntary caloric intake along with the rate of body weight gain dropped precipitously from normal and plateaued at 50 days, following which time she ingested 100±10 calories per kilogram of body weight per day (approximately 50% of normal). A similar drop and plateau at an approximate level followed complete ablation at 35 days in Animal P-790. As with the rate of body weight gain in Animal P-790, the first dose of I^{131} was followed by an incomplete and unsustained effect upon caloric intake. Intakes of nitrogen, calcium, and phosphorus bore a parallel relationship to calories in all animals, since the composition of formulae fed was constant.[18]

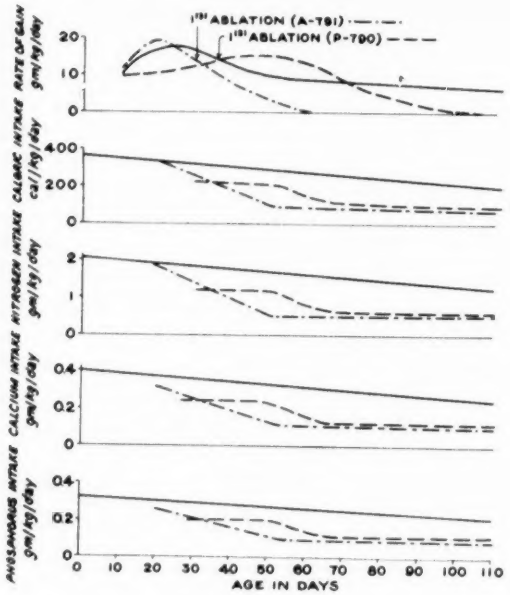

Fig. 4.—Rate of gain and nutrional intake of normal and athyroid macaque (A-791, P-790).

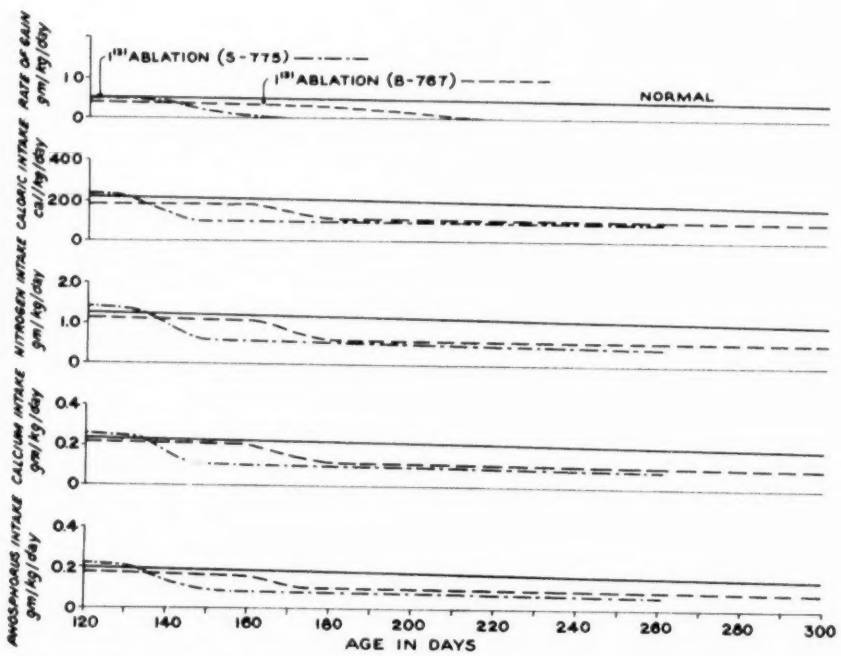

Fig. 5.—Rate of gain and nutritional intake in normal and athyroid macaques (S-775, B-767).

Figure 5 shows that voluntary food intakes of Animals B-767 and S-775 equaled the normal before they underwent I^{131} thyroid ablation at 162 and 122 days, respectively. Intakes then dropped until approximately 35 days after thyroid ablation, when they plateaued at approximately 50% of normal in a manner similar to that described for Animals A-791 and P-790. It is of interest that, once plateaued, caloric intake averaged 100 ± 10 calories per kilogram per day in all athyroid animals.

Quantities of nitrogen, calcium, and phosphorus retained by these animals during the period of plateaued growth were uniformly reduced from normal (Table 4).

TABLE 4.—*Nitrogen, Calcium, and Phosphorus Balance Data on Athyroid Infant Macaque Monkeys*

			Balance Data, Mg./Kg./Day								
			Calcium			Phosphorus			Nitrogen		
Animal	Weight, Gm.	Age, Days	Intake	Output	Balance	Intake	Output	Balance	Intake	Output	Balance
A-791.....	862	76-79	146	89	57	116	55	61	707	378	329
Female	818	95-98	110	49	61	90	36	54	552	270	282
P-790.....	1,143	141-144	164	150	14	133	114	19	737	331	406
Female	1,200	155-158	125	105	20	112	100	12	688	227	461
S-775.....	1,526	235-238	97	58	39	80	58	22	490	253	237
Female											
B-767.....	2,161	195-198	139	77	62	110	62	48	634	434	200
Female	2,208	205-208	136	107	29	108	68	40	626	320	306
Range average					40			36			317

TABLE 5.—*Creatine-Creatinine Excretion Data on Athyroid Infant Macaque Monkeys*

			Creatine-Creatinine Excretion, Mg./Kg./Day			
Animal	Sex	Age, Days	Creatine	Creatinine	Total	Ratio
A-791.....	Female	95-98	6.12	12.20	18.32	0.50
		116-119	3.00	13.10	16.10	0.23
		136-139	5.72	12.90	18.62	0.45
P-790.....	Female	141-144	2.04	10.20	12.24	0.20
		155-158	3.40	9.70	13.10	0.35
		164-167	6.62	11.90	18.52	0.55

The variation shown for different periods of study was no doubt influenced by the day-to-day fluctuation in the degree of myxedma and associated variation in tissue composition. Balance studies conducted over a similar age range in normal infants revealed average retentions (milligrams per kilogram per day) of 635 mg. of nitrogen, 135 mg. of calcium, and 117 mg. of phosphorus.[18]

Features of urinary creatine and creatinine excretion in these animals are shown in Table 5. Data shown in that table were gathered during six three-day periods of study conducted over a four- to five-week period on Animals A-791 and P-790.

Quantities of creatine and creatinine excreted were less than normal, though their ratio remained within the normal range. As with nitrogen, calcium, and phosphorus balance, subsequent to a period of *l*-thyroxine therapy sufficient in duration to increase the rate of growth to near normal, creatine and creatinine excretion returned to normal (7.5 ± 2.5 milligrams per kilogram per day and 20 ± 5 milligrams per kilogram per day, respectively).[19b]

SUMMARY AND CONCLUSIONS

In summary, within 30 to 45 days following I^{131} thyroid ablation, behavioral and physical features very much like those which characterize athyrosis in the human infant developed in the macaque infants studied. They coincidentally ceased growing and voluntarily restricted their caloric intakes and, in association, their nitrogen, calcium, and phosphorus retentions, and urinary excretion of creatine and creatinine were significantly reduced from normal.

Conclusions.—Behavioral, physical, growth, and metabolic features were investigated over a four-month period in two infant rhesus monkeys whose thyroids were ablated in the neonatal period and over a similar period in two others whose thyroids were ablated at 4 and 5 months, respectively.

I^{131} in amounts necessary to produce thyroid ablation caused neither significant change in the formed elements of blood nor hypoparathyroidism. At^{211}, though similarly concentrated by thyroid, precipitated a marked transient pancytopenia in two animals, after a relatively small dose was given. Its subsequent use was, therefore, abandoned in favor of I^{131}.

Characteristic behavioral and physical changes similar to those common to the human athyroid appeared 15 to 20 days following thyroid ablation and were marked by 40 to 45 days.

Coincident to the above, voluntary caloric intake dropped 50% to 100 ± 10 calories per kilogram per day, and growth ceased.

Nitrogen, calcium, and phosphorus retention was greatly reduced from normal and fluctuated with the varying degree of myxedema. Similarly, urinary creatine and creatinine excretion averaged approximately 50% of normal.

Mr. Ralph Sweet and his staff provided the illustrations included in this report; Miss Margaret Cole and Miss Mary Gaylord gave technical assistance, and Dr. Kenneth G. Scott, Director, The Radioactivity Research Center, furnished technical supervision and suggestions.

CONGENITAL SARCOMA OF THE FOOT

Case Report and Review of Literature

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AND
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EVANSTON, ILL.

CONGENITAL sarcoma is rare and has usually been reported as single cases. A total of 35 proved cases has been recorded in the world literature, 17 of which occurred in one of the extremities.

In a comprehensive review of congenital malignant lesions, Wells [1] excluded several reported as certain cases and listed them as probable or possible because of insufficient evidence regarding the time of onset or confirmation of the microscopic examination. He found that congenital sarcoma occurred as frequently as all other types of congenital tumors, such as neuroblastoma of the adrenal and sympathetic nervous system (sympatheticoblastoma), retinoblastoma, mixed renal tumors, and hemangioendothelioma of the liver. While the predominant type of tumor occurring in the extremity was fibrosarcoma, cases of osteogenic sarcoma were recorded and one case each of angiosarcoma and liposarcoma.

In 3,000 necropsies on newborn infants, Wells [1] did not find an instance of congenital sarcoma. In his series, only four congenital malignant neoplasms, all neuroblastomas, were found, three in the adrenal and one involving the sympathetic nervous system. He believed that congenital sarcomas have origin, growth, and behavior characteristics similar to the adult type.

Only one case of congenital soft tissue sarcoma was encountered in a 22-year period (1917 to 1938) at Memorial Hospital, New York City, which is devoted to the treatment of cancer and allied diseases.[2]

Potter,[3] in an extensive experience with pathology of the fetus and newborn, at the Chicago Lying-in Hospital of the University of Chicago, encountered only one case which she regarded as congenital sarcoma.

Chief of Orthopedic Surgery (Dr. Fahey) and Resident in Surgery (Dr. Bollinger), Department of Orthopedic Surgery, St. Francis Hospital.

1. Wells, H. G.: Occurrence and Significance of Congenital Malignant Neoplasms, Arch. Path. **30**:535-601, 1940.

2. Pack, G. T., and Anglem, T. J.: Tumor of the Soft Somatic Tissues in Infancy and Childhood, J. Pediat. **15**:372-400, 1939.

3. Potter, E. L.: Pathology of the Fetus and Newborn, Chicago, Year Book Publishers, Inc., 1952, pp. 142-171.

On the basis of reported cases, early amputation is the treatment of choice. Of the 17 cases of congenital sarcoma that occurred in the extremities, there was no record of follow-up treatment in 4 cases. One patient died at 3 months of age untreated, and two died postoperatively. One died 16 months following amputation, and one patient was living 11 months following amputation. Six patients were living and well from 2 to 10 years following surgery. One of these patients was treated by excision, another by x-ray, and the remainder by amputation. There were only two patients said to have recovered; one was treated by surgery and the other by surgery and x-ray, but the length of survival was not mentioned.

Because of the rarity of congenital sarcoma, a case report appears justified. Only two other cases have been reported involving the foot.

REPORT OF A CASE

A 3-month-old infant was seen because of a large mass on the inner aspect of the left foot (Fig. 1). On the second day after birth, a small protuberant tumor, the size of the tip of the

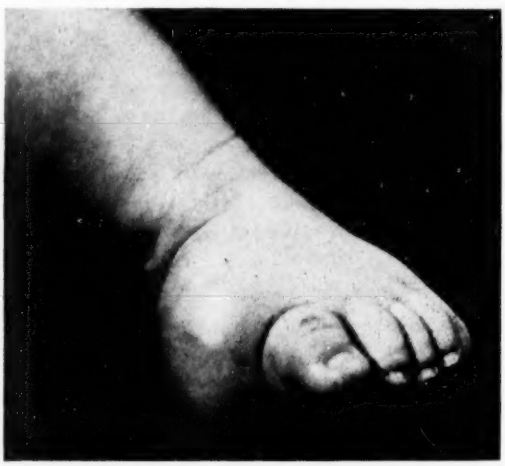

Fig. 1.—Appearance of the tumor on the medial aspect of the foot at the age of 3 months. The tense shiny skin covering the tumor may be noted.

finger, was noted on the inner side of the foot, and it had grown rapidly. When one of us (J. J. F.) first saw the child, at 3 months of age, the tumor had protruded medially from the inner side of the foot from the base of the big toe to the heel, enlarging the dorsal and ventral surfaces of the foot. The overlying skin was tense, shiny, and moveable, and there were many enlarged veins. While the tumor appeared to be distinct from the foot, the foot itself appeared thickened. There were no palpable glands in the extremity or the groin. The heart and lungs were normal. Findings on x-rays of the chest were normal. The red blood cell count was 4,050,000, and the white blood cell count was 7,700. The urine was normal except for a trace of albumin. X-ray examination (Fig. 2) of the foot showed a soft tissue mass on the medial aspect of the foot. There was widening between the first and second metatarsals and the respective phalanges. There was a concavity deformity of the volar surface of the first metatarsal and the inner side of the second metatarsal which had resulted from tumor pressure.

Permission was obtained only for biopsy and local excision. At surgery, a well-encapsulated tumor 2 by 4.5 by 5 cm. (Fig. 3) was found attached only at one point near the distal portion of the first metatarsal. The surface of the tumor was pale, opaque, and compact.

Microscopic examination showed the surface of the tumor to be covered by a rather thick fibrous tissue capsule which had been invaded by tumor cells. Under low power (Fig. 4), the cells were closely packed and arranged in whorls. Under high power (Fig. 5), the cells had indefinite boundaries and pleomorphic nuclei which varied from small, round nuclei to a large and

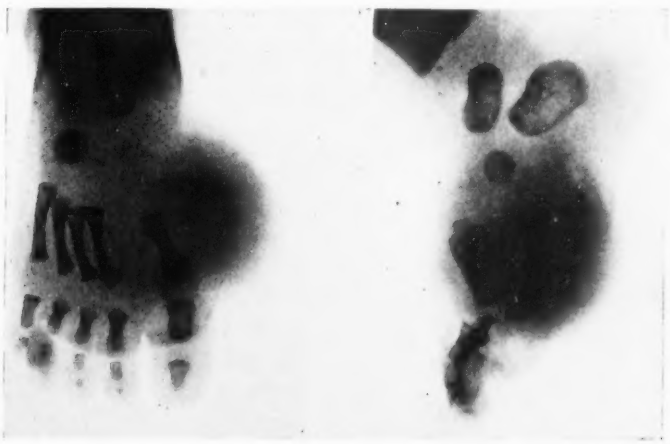

Fig. 2.—X-ray shows the soft tissue shadow of the tumor. There is widening between the first and second metatarsals and the respective phalanges. The volar surface of the first metatarsal and the inner side of the second metatarsal showed concave deformity resulting from tumor pressure.

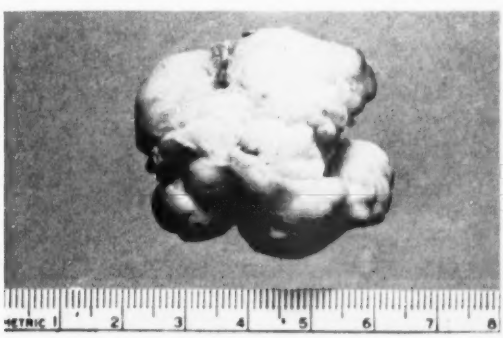

Fig. 3.—The gross specimen consists of an irregular soft lobulated mass (2 by 4.5 by 5 cm.) well encapsulated except in one small region.

lobulated configuration. The cytoplasm in many of the large cells was either spongy or granular. Many mitotic figures were present. The microscopic picture was consistent with fibrosarcoma.

The child was readmitted four months later with a local recurrence, and the appearance of the foot simulated very closely its appearance before the first operation. There were no palpable glands in the groin, and findings on x-rays of the chest were normal. The child appeared in good health, and no evidence of metastasis could be found.

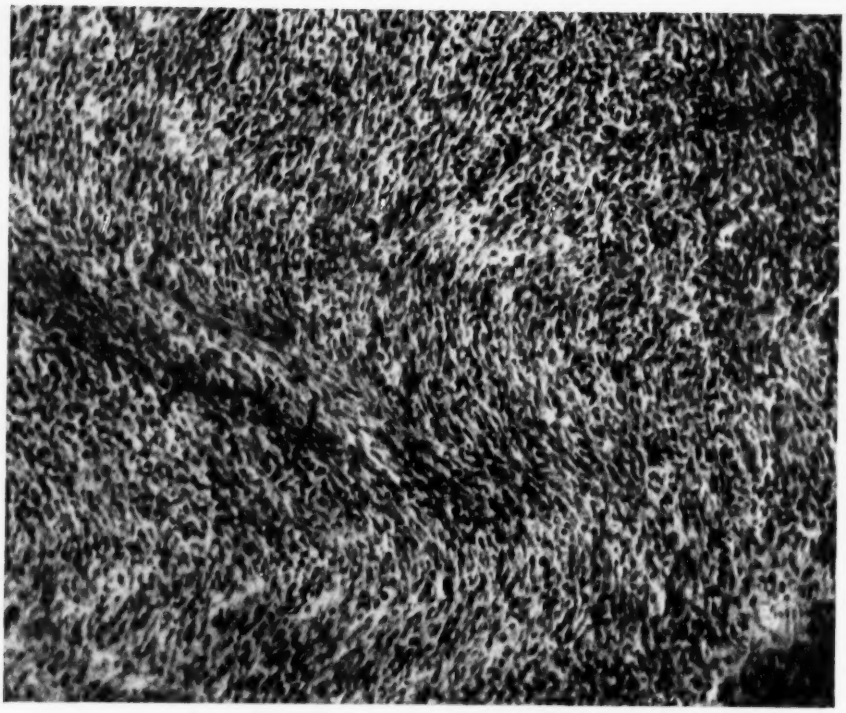

Fig. 4.—Microscopic examination (low power) reveals the closely packed cells giving a fasciculated arrangement.

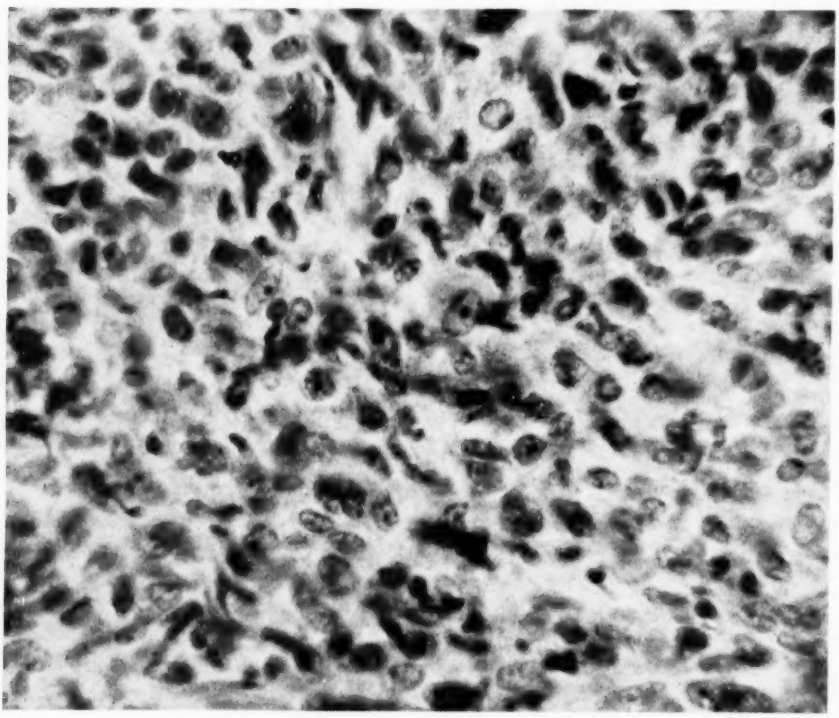

Fig. 5.—Microscopic examination (high power) shows cells with indefinite boundaries and pleomorphic nuclei. The latter vary from small deeply stained nuclei to large lobulated nuclei. Cytoplasm in large cells is spongy to granular. Many mitotic figures are present.

Consent for amputation was finally obtained and carried out in the upper tibia. Approximately seven months following amputation, she became anemic; rales developed in the chest, and she died nine months following amputation. A postmortem examination was not done.

SUMMARY

Congenital sarcoma is rare. One case is added to the two cases previously reported in the literature, originating in the foot.

Based on the reports in the literature, early amputation offers the best chance of success. When a tumor of unknown origin is identified in the newborn, a period of watchful waiting, coupled with the consent for only local excision later, may preclude the only possibility of survival.

PRIMARY MYOCARDIAL DISEASE IN INFANCY AND CHILDHOOD

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RHEUMATISM heart disease and congenital heart disease account for the vas majority of cardiac disorders in infancy and childhood.[1] Within the past few years, a number of patients have been seen at the Children's Medical Center with severe heart disease of a type which could not be fitted into either of the two groups mentioned above. They all had the following features in common: (1) cardiomegaly; (2) absence of significant murmurs; (3) electrocardiographic abnormalities, and (4) normal blood pressure.

These children did not appear to have rheumatic heart disease because of their very early age and the absence of the major and minor manifestations of rheumatic fever.[2] Congenital heart disease in the conventional sense (i. e., anomalies of the great vessels, valves, and septa) was excluded by the absence of murmurs, abnormal blood pressure, or evidence of shunts in either direction. Paroxysmal auricular tachycardia was excluded by the electrocardiographic finding of a sinus pacemaker and the persistence of cardiomegaly. Pericarditis was considered unlikely in the absence of electrocardiographic, roentgenologic, or clinical evidence of this condition. Furthermore, the course of the disease in these patients, as well as their response to treatment, did not suggest pericarditis.

The clinical picture presented by this group, plus autopsy experience on some of these patients, led one to believe that this syndrome was a manifestation of primary disease of the myocardium. No patient was included in this group who was known to have any of the communicable diseases (diphtheria, poliomyelitis, etc.) or diseases of the central nervous system (Friedreich's ataxia, etc.) which may be associated with a myocarditis. Four patients are included, however, in whom a differentiation between "primary myocardial disease" and myocarditis secondary to pneumonitis was practically impossible. These four patients had physical findings of rales and harsh breath sounds with chest films equally suggestive of inflammation or congestion.

Supported by a grant from the Massachusetts Heart Association.

Presented in part before the Radiological Society of North America, Cincinnati, on Dec. 12, 1952.

From the Departments of Pediatrics and Radiology, Harvard Medical School, and from the Children's Medical Center, Boston.

1. Gibson, S., in Practice of Pediatrics, edited by J. Brennemann, Hagerstown, Md., W. F. Prior Company, Inc., 1948, p. 4385.

2. Jones, T. D.: The Diagnosis of Rheumatic Fever, J. A. M. A. **126**:481, 1944.

The purpose of this study is to emphasize a clinical syndrome not generally well recognized, to stress its relative frequency, to review the literature on the subject, and to delineate its origin, course, and treatment.

MATERIAL

Forty-five patients with the clinical picture of myocardial disease as defined above constitute the material for this study. Twenty-six of these have died and come to autopsy. On this group, both pathological and clinical data are available. On the surviving 19 patients, detailed clinical data are available.

RESULTS

Autopsy Findings.—The five pathological entities which produced this clinical syndrome, and their relative frequency, may be seen in Table 1.[3] It is evident that glycogen-storage disease of the heart, aberrant left coronary artery, and medial necrosis of the coronary arteries are rare. Most of the deaths were due either to subendocardial sclerosis or to idiopathic myocarditis.

Clinical Findings.—Generally speaking, the onset is quite rapid without prodromal symptoms. Respiratory distress with intermittent cyanosis, and occasionally

TABLE 1.—*Autopsy Findings in Twenty-Six Cases of Primary Myocardial Disease*

Disease	No. Cases
Glycogen-storage disease of the heart	3*
Aberrant left coronary artery	1†
Medial necrosis of the coronary arteries	2‡
Idiopathic myocarditis	10‡
Subendocardial sclerosis	10§
Total	26

* Two of these cases previously reported by Landing and Bangle.[3a]
† Previously reported by Craig.[3b]
‡ Three of these cases previously reported by House.[3c]
§ Seven of these cases previously reported by Craig.[3b]

with cough, is usually the dominating complaint. Anorexia, vomiting, and failure to gain are frequently present. In a few cases, malaise and irritability are the only symptoms. In these patients, the nature of the illness is usually first suggested by tachycardia or a chest film showing cardiac enlargement.

In most instances, the physical examination reveals the patient to be acutely and gravely ill with tachypnea and tachycardia out of proportion to the temperature. Pallor and sweating are usually present, and mild to moderate retraction is common. Cyanosis is seen in the majority of cases. Gallop rhythm, heart sounds of poor quality, and abnormal breath sounds are frequently heard. Evidence of congestive heart failure (hepatomegaly, distended neck veins, rales) is present, and often severe, in most of the patients. Peripheral edema is unusual. A few patients are less seriously ill and show only cardiomegaly by physical examination.

In an attempt to discover a basis for differentiation of these five conditions in living patients, the significant clinical features presented by the autopsied patients

3. (*a*) Landing, B. H., and Bangle, R., Jr.: Glycogen Storage Disease: I. Familial Cardiac Glycogen Storage Disease; Report of 2 Cases and Discussion of Relation to Other Forms of Abnormal Glycogen Deposition, Bull. Internat. A. M. Mus. **31**:84, 1950. (*b*) Craig, J. M.: Congenital Endocardial Sclerosis, ibid. **30**:15, 1949. (*c*) House, R. K.: Diffuse Interstitial Myocarditis in Children, Am. J. Path. **24**:1235, 1948.

TABLE 2.—*Clinical Findings in Twenty-Six Autopsied Patients with Primary Myocardial Disease*

Disease	Patient	Sex	Age at Onset	Family History	Chief Complaint	Dyspnea	Cyanosis	Edema	Temperature, F. (Maximal)	Pulse	Liver (Maximal)	Heart Sounds
Glycogen-storage disease of the heart	J. F. McL.	M	2 mo.	2 brothers with disease	Vomiting; abscesses; lassitude	No	No	No	99.2	140	3 cm.	Negative
	P. J. McL.	M	1 mo.	2 brothers with disease	Large heart	No	No	No	101.5	110	1 cm.	Negative
	B. McL.	M	1 mo.	2 brothers with disease	Dyspnea; cough; furuncles	Yes	No	No	.....	...	3 cm.	Gallop
Aberrant left coronary.....	R. B.	M	2 mo.	Negative	"Attacks" of dyspnea and cyanosis	Yes	Yes	No	102.5	Rapid	3 cm.	Negative
Medial necrosis of the coronary arteries	L. M. B.	F	3 wk.	Negative	Pallor; dyspnea; cyanosis	Yes	Yes	No	101.5	116	Iliac crest	Negative
	J. G. L.	M	2 mo.	1 sibling (Mongol) died 1st mo.; 1 died of pneumonia (1 mo.)	Irritability; anorexia	No	Yes	No	96.4	140	3 cm.	Negative
Idiopathic myocarditis	B. C.	F	3 wk.	Negative	Dyspnea; cyanosis; vomiting	Yes	Yes	No	100	...	Iliac crest	Muffled
	D. D.	F	10 mo.	Negative	Dyspnea; cough	Yes	Yes	No	104	180	4 cm.	distant Negative
	R. D'A.	M	1 yr.	Negative	"Cold sweats"; dyspnea	Yes	No	No	102.5	160	4 cm.	Weak; poor quality
	B. G. H.	F	1 day	Mother has RHD	Cyanosis	Yes	Yes	No	.....	120	7 cm.	Muffled
	J. K.	F	8 mo.	Negative	Listlessness; "sudden death"	No	Yes	No	.....	...	5 cm.	.....
	J. G.	F	6½ yr.	Negative	Vomiting; listlessness; pallor	No	No	No	99.5	140	6 cm.	Gallop
	R. M.	M	10 yr.	Strong for RHD and CHD	Abdominal and back pain; vomiting	Yes	No	Yes	104	120	8 cm.	Gallop
	A. H.	M	3 mo.	Negative	Wheezing; labored respirations; weakness	Yes	Yes	No	104.6	160	4 cm.	Negative
	J. B.	M	1 mo.	Negative	Dyspnea; cyanosis	Yes	Yes	No	99.8	200	3 cm.	Negative
	P. L. M.	F	2 mo.	Negative	Dyspnea; lethargy; anorexia	Yes	Yes	No	100.8	170	4 cm.	Gallop
Subendocardial sclerosis	J. K.	F	5 mo.	Negative	Fever; sweating	Yes	Yes	Yes	101	140	7 cm.	Negative
	L. B.	F	7 mo.	Negative	Dyspnea	Yes	No	No	103.5	150	5 cm.	Distant
	S. D.	F	4 mo.	Negative	Dyspnea; collapse	Yes	Yes	No	102	...	4 cm.	Poor quality
	B. G. H.	F	24 hr.	Negative	Cyanosis; heart block	No	Yes	No	101	58	1 cm.	Negative
	C. A. B.	F	4 mo.	Uncle had CHD	Dyspnea; cyanosis; vomiting	Yes	Yes	No	100.5	210	Iliac crest	Negative
	M. L. M.	F	3 mo.	Negative	Cough; fever	No	Yes	No	104	140	0	Negative
	M. C.	F	4 mo.	Negative	Dyspnea	Yes	No	No	101	185	4 cm.	Negative
	T. V.	M	3 wk.	Negative	Dyspnea; cough	Yes	Yes	No	104	125	2 cm.	Negative
	M. L. T.	F	1 wk.	Negative	Jaundice	No	Yes	No	97	110	4 cm.	Negative
	R. St. P.	M	4 mo.	Negative	Irritability; dyspnea; cough	Yes	No	No	98	86	6 cm.	Gallop

TABLE 3.—*Clinical Findings in Nineteen Living Patients with Primary Myocardial Disease*

Patient	Sex	Age at Onset	Family History	Chief Complaint	Dyspnea	Cyanosis	Edema	Chest Deformity	Temperature, F. (Maximal)	Pulse	Liver (Maximal)	Heart Sounds
N. L.	M	7 yr.	Negative	Anorexia; listlessness	Yes	Yes	Yes	Yes	100.4	100	7 cm.	Negative
J. P.	M	1 10/12 yr.	Negative	Dyspnea; wheezing	Yes	No	No	Yes	103	150	5 cm.	Negative
E. E.	M	8 mo.	Negative	"Colds"; large heart	No	No	No	No	101	150	0	Distant; poor quality
C. B.	M	2 5/12 yr.	Negative	Dyspnea; listlessness	Yes	No	No	Yes	101	120	9 cm.	Weak, gallop
S. F.	F	6 mo.	Negative	Dyspnea; restlessness	Yes	Yes	No	No	101.2	160	7 cm.	Poor quality
W. F. H.	M	11 days	Negative	Dyspnea; cyanosis	Yes	Yes	No	No	100.4	150	3 cm.	Mushy
L. P.	F	1 yr.	Negative	Dyspnea; irritability	Yes	No	No	No	100.8	130	5 cm.	Poor quality
C. M.	M	4 mo.	1 sibling was "blue baby"	Dyspnea; anorexia	Yes	No	No	No	100	130	3 cm.	Negative
P. C.	F	7 days	Negative	Attacks of cyanosis	No	Yes	No	No	99.4	120	1 cm.	Negative
M. H.	F	3 wk.	Negative	Dyspnea; cyanosis	Yes	Yes	Yes	....	99.8	140	5.5 cm.	Muffled
M. D.	F	1 mo.	Negative	Severe sweating; poor feeder	No	No	No	Yes	99.4	150	3.0 cm.	Negative
C. L.	F	6 yr.	Negative	Fever; malaise	Yes	No	No	No	105.2	150	6 cm.	Muffled gallop
W. B.	M	2 4/12 yr.	Positive for ichthyosis	Dyspnea; listlessness	Yes	No	Yes	No	99.4	153	6.5 cm.	Gallop, poor quality
B. S.	F	6 mo.	.....	Dyspnea; cyanosis	Yes	Yes	No	No	100	150	4.5 cm.	Negative
S. E.	F	Birth	1 sibling with extrophy of bladder	Cyanosis; poor feeder	No	Yes	No	No	99.2	160	4 cm.	Negative
I. L.	M	5 1/12 yr.	Negative	"Rapid heart"	No	No	No	No	100.4	200	4 cm.	Poor quality
A. L. L.	F	15 mo.	Negative	Edema	No	No	Yes	No	100.2	150	7 cm.	Gallop
R. G.	F	Birth	Negative	Dyspnea	Yes	No	No	No	100	158	5 cm.	Mushy
D. A.	M	3 mo.	Negative	Dyspnea; cyanosis	Yes	Yes	Yes	Yes	99.2	160	5 cm.	Gallop

are recorded in Table 2. Table 3 contains similar data for the living patients. The findings in both of these tables are summarized in Table 4. The fact that all of the patients had cardiac enlargement and none of them had significant murmurs (beyond Grade II in intensity, or "organic" in quality) is not represented in these tables since it is part of the definition of this clinical syndrome.

It may be seen that the clinical picture presented by all five of these conditions is very similar. A few observations are recorded, however, that may help in making a differential diagnosis. These include an age at onset of symptoms of less than three months in glycogen-storage disease of the heart and the coronary artery diseases. In contrast, the age at onset of the living patients and those with idiopathic

TABLE 4.—*Summary of the Clinical Findings in Forty-Five Patients with Primary Myocardial Disease*

Disease	No. Cases	Sex	Age at Onset: 6 Mo. or Less	Age at Onset: Over 6 Mo.	Positive Family History	Complaint Chief	Dyspnea	Cyanosis	Edema	Fever (Maximal) Over 101 F.	Pulse Rate (Maximal) Over 140 Min.	Hepatomegaly (Over 3 Cm.)	Abnormal* Heart Sounds
Glycogen-storage disease of the heart	3	3 M	3	0	3	Cardiomegaly, dyspnea, vomiting	1	0	0	1	0	0	1
Aberrant left coronary artery	1	1 M	1	0	0	"Attacks" of dyspnea, cyanosis, and perspiration	1	1	0	1	1	0	0
Medial necrosis of the coronary arteries	2	1 M 1 F	2	0	0	Pallor, dyspnea, cyanosis	1	2	0	1	0	1	0
Idiopathic myocarditis	10	4 M 6 F	5	5†	1	Dyspnea, cyanosis, cough, anorexia	8	7	1	4	5	9	6
Subendocardial sclerosis	10	2 M 8 F	9‡	1	1	Dyspnea, cyanosis, cough, anorexia	7	7	1	4	3	7	3
Living patients	19	9 M 10 F	10	9§	1	Dyspnea, cyanosis, cough, anorexia	13	8	5	3	13	14	12

* Gallop rhythm or sounds of poor quality.
† One patient 6½ yr. old; another 10 yr. old.
‡ Six of nine 3 mo. of age or older.
§ Eight patients 1 yr. of age or older; three patients older than 5 yr.

myocarditis was frequently much later. Subendocardial sclerosis occupied an intermediate position. Cyanosis was a common feature in all of the diseases. The strong family history in cardiac glycogen storage disease is striking. Abnormal heart sounds were present in more than half of the living patients and those with idiopathic myocarditis. Congestive cardiac failure, as evidenced by hepatomegaly, was present in the vast majority of the living patients and in those with idiopathic myocarditis and subendocardial sclerosis, but was rarely seen in any of the other conditions.

Laboratory findings are not tabulated, since no points of differential value or striking interest were discovered.

Roentgenographic Findings.—X-ray examination was performed on the patient with an aberrant left coronary artery, both patients with medial necrosis of the

coronary arteries, and two of the three patients with glycogen-storage disease of the heart. Eight of the 10 patients with a pathological diagnosis of idiopathic myocarditis were similarly examined, as were 7 of the 10 patients with subendocardial sclerosis. All of the living patients have had complete roentgenographic studies.

The roentgen pictures of all the patients (living and dead) have presented just as great a similarity as did their clinical features. The heart has been enlarged in every instance and usually to a marked, or even an extreme, degree. This enlargement is generalized, involving all chambers of the heart (Figs. 1 and 2). The generalized aspect of the enlargement is of great diagnostic importance since other types of cardiac disease in the pediatric age group usually show some prefer-

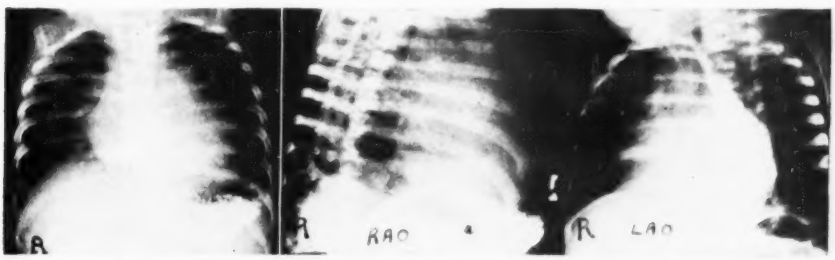

Fig. 1.—B. L. S., a 7-month-old white girl, admitted because of cough and rapid respirations. No murmur; gallop rhythm. Typical electrocardiographic changes (See Fig. 5*A*). Posteroanterior and both antero-oblique films of the chest show marked generalized cardiomegaly with pulmonary congestion.

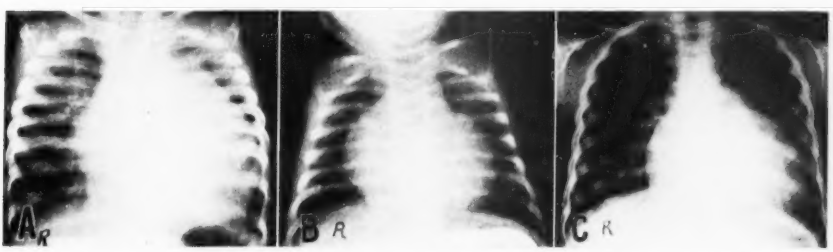

Fig. 2.—Posteroanterior films of the chest of three patients with primary myocardial disease. All show marked cardiac enlargement. *A*, E. E., a one-year-old white boy, admitted because of an enlarged heart. No murmur; heart sounds of poor quality. Electrocardiogram showed left ventricular hypertrophy with T-wave changes. Note marked pulmonary congestion. *B*, S. E., a 2-month-old white girl, admitted with cough and failure to gain. No murmur. Typical electrocardiographic changes (See Fig. 4). Note absence of pulmonary congestion. *C*, L. L., a 5-year-old white boy, admitted with tachycardia, cough, and malaise. No murmur; gallop rhythm. Typical electrocardiographic changes (See Fig. 6).

ential chamber predominance. The only exception to this is pericardial effusion, which is sometimes difficult to differentiate roentgenologically, for in both instances the amplitude of pulsation as seen at fluoroscopy is markedly diminished.

Usually the silhouette of the heart is globular in shape. It is not unusual for the heart to touch the left lateral chest wall because of its massive size. In fact, at times the entire left chest is practically filled with the cardiac shadow. This frequently leads to pressure upon bronchi, with resulting poor air exchange and even collapse of portions of the left lung.

Except for evidence of engorgement in half of the cases, the pulmonary vessels are not remarkable. The main pulmonary artery and its major branches are usually obscured by the shadow of the heart itself. The absence of an x-ray appearance of increased or decreased pulmonary blood flow is very valuable evidence against left-to-right shunts and pulmonic stenosis, respectively.

In four instances, the lung fields have shown areas of decreased radiolucency which may have been due to either congestion or inflammation. Four other patients demonstrated areas of atelectasis varying in size from numerous plate-like streaks to collapse of entire lobes. One patient showed a definite pleural effusion, and 18 patients out of a total of 35 in whom this factor could be evaluated showed definite evidence of pulmonary congestion.

Twelve of the 19 living children have shown a definite decrease in the size of the heart following treatment and the passage of time. Generally speaking, this decrease in size has occurred slowly but steadily. In one instance, the heart eventually returned to normal size; in another, almost normal size has been attained (Fig. 3).

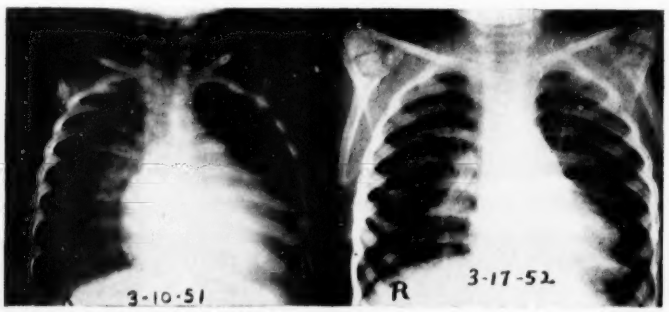

Fig. 3.—L. P., a 13-year-old white girl, admitted with malaise, irritability, and dyspnea. Grade I soft apical systolic murmur. Heart sounds of poor quality. Neck veins distended and the liver extended 5 cm. below the right costal margin in the nipple line. Electrocardiogram showed left ventricular hypertrophy with T-wave changes compatible with myocardial damage. She was given digitalis with complete disappearance of symptoms. Symptoms have not returned despite 18 months of full activity. Posteroanterior films of the chest show marked generalized cardiomegaly on admission (March 10, 1951) which largely disappeared over a period of one year (March 17, 1952).

Nothing was discovered in a study of the roentgenographic data on these patients that would aid in separating them according to cause.

Electrocardiographic Findings.—Fifteen out of the 26 autopsied patients were examined by electrocardiography. Four of these had complete examinations, including unipolar leads. The other 11 were only examined by standard limb leads. The 11 patients that did not have electrocardiograms are included in this group, since their clinical and pathological findings indicated that they properly belonged in this category. All of the living patients have had complete examinations, including unipolar chest leads and unipolar limb leads.

Regardless of cause, the electrocardiograms of the patients who came to autopsy were quite similar, as were the clinical features and x-ray appearances.

In 13 of the 15 autopsied patients examined, definite T-wave changes in the form of flattening or inversion were present. These changes involved T_{1-2} and T in

$V_{5\,6}$ when unipolar chest leads had been taken. Six patients in this group showed tall, peaked P waves, and three of the four patients with complete electrocardiographic examinations demonstrated left ventricular hypertrophy.

Only minor and inconstant ST changes were noted. ST depression would theoretically be expected in subendocardial sclerosis, since the pathology has a location similar to that of subendocardial infarctions,[4] but no such changes were observed in either the dead or living patients.

Two of the patients in the autopsied group showed complete heart block, and two others showed prolonged atrioventricular conduction. One patient demonstrated very low voltage in his electrocardiogram.

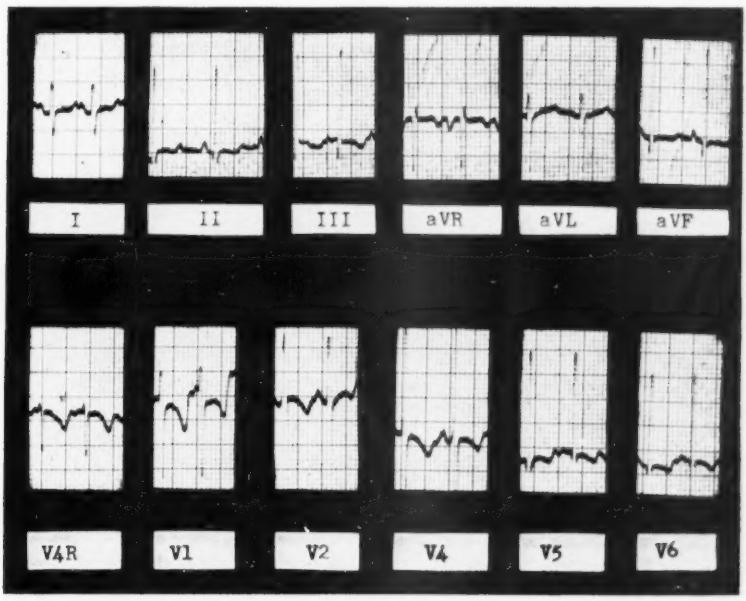

Fig. 4.—S. E., a 2-month-old girl, admitted because of cough and failure to gain. X-ray showed marked generalized cardiomegaly. No murmur. Note diphasic T_1, T_2; inverted T in V_4, V_5, V_6; deep S in V_1R and tall R in V_5. She improved on symtomatic therapy. Seven months later, T waves were upright, and she was without symptoms. Cardiomegaly remained unchanged.

Of the 19 living patients, 14 showed left ventricular hypertrophy, 2, right ventricular hypertrophy, and 3 showed no significant ventricular preponderance. Nine of the 19 patients showed evidence of auricular hypertrophy.

Significant T-wave changes in the form of flattening and inversion in Leads I and II and the left-sided unipolar chest leads were present in 18 of the 19 patients. The one patient that failed to show T-wave changes was one of the patients with right ventricular hypertrophy and marked peripheral edema. Her tracing demonstrated extremely low voltage; consequently, her T waves were essentially flat.

4. Levine, H. D., and Ford, R. V.: Subendocardial Infarction: Report of 6 Cases and Critical Survey of the Literature, Circulation **1**:246, 1950.

Two patients showed prolonged P-R intervals. Three patients demonstrated paroxysmal supraventricular tachycardia at least once. One of these had a Wolff-Parkinson-White syndrome.

In summary, the electrocardiographic changes usually seen in this myocardial syndrome are left ventricular hypertrophy with T-wave changes of myocardial damage. Auricular hypertrophy and rhythm and conduction disturbances are not unusual (Figs. 4 and 5; all electrocardiograms reproduced were taken while the patient was not receiving digitalis). It should be pointed out that conventional R-S progression in an infant, who under normal conditions would show right ventricular hypertrophy, is, in fact, indicative of hypertrophy of the left ventricle. These T-wave

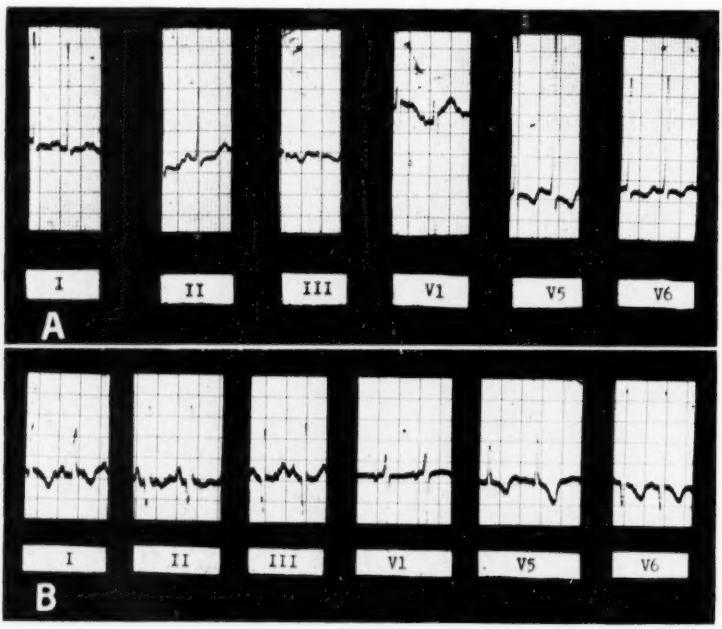

Fig. 5.—*A*, B. L. S., 7-month-old girl, admitted because of cough and rapid respirations. X-ray showed marked generalized cardiomegaly. No murmurs; gallop rhythm. On digitoxin she became asymptomatic and remained so, although her cardiomegaly and electrocardiographic abnormalities have persisted. Note diphasic T_1, T_2; inverted TV_5, TV_6; deep SV_1 and tall RV_6. *B*, C. M., 7-month-old boy, admitted with pallor, tachypnea, and tachycardia. X-ray showed marked generalized cardiomegaly. No murmurs, but definite evidence of congestive failure. On digitoxin he rapidly improved and became free of symptoms. Although cardiomegaly and electrocardiographic changes have persisted he has remained active and without symptoms on digitoxin. Note tall peaked P_2; inverted T_1, T_2, TV_5, TV_6; deep SV_1, tall RV_6.

changes in infants have been thought to be diagnostic of an aberrant left coronary artery because such changes, as well as low voltage, have been reported [5] in this condition; however, such changes are only indicative of myocardial damage regardless of cause.

5. Bland, E. F.; White, P. D., and Garland, J.: Congenital Anomalies of Coronary Arteries: Report of an Unusual Case Associated with Cardiac Hypertrophy, Am. Heart J. **8**:787, 1933. Taussig, H. B.: Congenital Malformations of the Heart, New York, Commonwealth Fund, 1947, p. 320.

T-wave changes have been observed to disappear in eight of these patients over a period of months (Fig. 6). In several other patients, the electrocardiographic abnormalities have become less marked.

Response to Treatment.—A good number of the autopsied patients were moribund on admission and died within a very few hours of arrival. Consequently, therapeutic measures could not be appraised in these individuals. All of these patients received oxygen and various stimulants without definite effect. Because of the short duration of hospitalization in many patients and because of failure to appreciate the true nature of the illness in others, only seven patients in the autop-

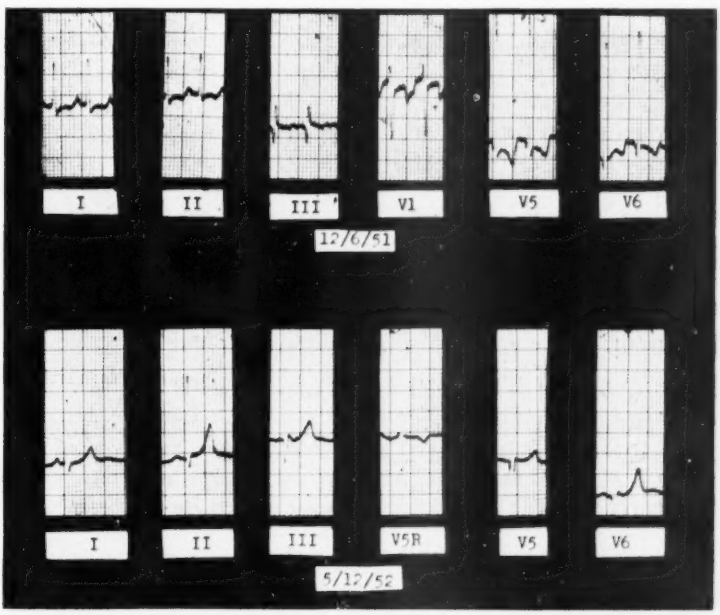

Fig. 6.—L. L., 5-year-old boy, admitted with tachycardia, cough, and malaise. X-ray showed marked generalized cardiomegaly. No murmur; gallop rhythm; marked hepatomegaly; definite congestive failure. Digitoxin was given with good steady response. At present patient is without symptoms on full activity. He no longer takes digitoxin. Cardiomegaly persists. Note that T-waves have become upright since admission (Dec. 6, 1951).

sied group received digitalis. Three of these, all with idiopathic myocarditis, showed definite improvement for a time, only to deteriorate later and die.

Thirteen of the 19 living patients received digitalis. Five of the six remaining patients in this group did not receive any glycoside because they did not show sufficient evidence of congestive failure. A roentgen finding of cardiomegaly or difficulty with feeding, etc., prompted their hospital admission. The remaining patient in congestive failure who did not receive digitalis was treated with cortisone with eventual disappearance of symptoms.

Digitalis has been given in the form of digitoxin by mouth except in emergencies where the intravenous route has been employed. Between 0.02 mg. and 0.025 mg. per pound (0.5 kg.) of body weight has been used as a digitalizing dose. This has

been given over a period of 24 hours, or more rapidly if the gravity of the situation warranted. One-tenth of the digitalizing dose generally has been administered for daily maintenance.

The response of the majority of the living patients to digitalis was excellent. In 10 of the 13 children treated with the glycoside, signs of congestive failure disappeared. Coincident with the marked clinical improvement was the appearance of electrocardiographic evidence of digitalis intoxication in seven patients. The striking sensitivity of these patients to digitalis, as exemplified by both their good therapeutic response and frequent signs of toxicity, has been discussed in detail in another publication.[6]

Only three of the 13 patients treated with digitalis failed to improve on this therapy alone. These three responded satisfactorily, however, to mercurial diuretics. This small group of three patients presented a somewhat different picture from the other patients and resembled constrictive pericarditis, with marked peripheral edema and electrocardiograms showing low voltage and/or right ventricular hypertrophy. The diagnosis of constrictive pericarditis was ruled out by their subsequent course and by cardiac catheterization in two patients and by surgical exploration in the third.

General supportive measures in the form of oxygen, antibiotics, etc., were given as indicated to all patients in both groups.

The experience with the living patients indicates that the prognosis in this syndrome may be fairly good. Almost all of the surviving patients eventually have become free of symptoms, with or without digitalis. Those patients who improved after digitalization have been maintained on the glycoside for several months, even years. Termination of digitalis administration has, at times, resulted in reappearance of the symptoms of congestive failure. When digitalis can be safely discontinued must be determined in every case by trial periods without the drug.

In approximately half of the patients, the T-wave changes and left ventricular hypertrophy disappeared or became less marked. In an approximately equal number of instances, the heart became smaller by x-ray, occasionally reaching normal, or near normal, size. The patients that show a decrease in cardiomegaly are not, in every instance, those that also show less marked electrocardiographic abnormalities. In nine patients, cardiac enlargement and electrocardiographic changes have persisted as long as two years despite normal exercise tolerance and disappearance of symptoms. The long-term prognosis for this group is unknown at this time.

COMMENT

Diseases of the myocardium causing the clinical syndrome described above (cardiomegaly, absence of significant murmurs, electrocardiographic abnormalities, and normal blood pressure) are not uncommon. Over 20 such patients have been studied at our institution during the last three years.

Since this is primarily a clinical study, no attempt has been made to review the pathology of these conditions except insofar as such information aids in their identificaton in the living patient. The literature contains numerous excellent patho-

6. Nadas, A. S.; Rudolph, A. M., and Reinhold, J. D. L.: The Use of Digitalis in Infants and Children: A Clinical Study of Patients in Congestive Heart Failure, New England J. Med. **248**:98, 1953.

logical studies of these entities, many of which are referred to in the succeeding sections. In spite of the fact that the five pathological entities underlying this clinical syndrome of "primary myocardial disease" are difficult and often impossible to distinguish in the living patient, certain clinical features of differential value have become apparent from this study, as well as from a perusal of the literature. These matters can best be discussed in separate paragraphs for each of the pathological processes.

Glycogen-Storage Disease of the Heart.—Less than 40 authentic cases of this condition have been reported since its original description in 1932.[7] The pathological picture, consisting of abnormally large glycogen deposits in the heart and skeletal muscles, was recently reviewed by Landing and Bangle.[8a] These authors, as well as others,[8] stress the fundamental difference between this condition and von Gierke's disease, where large amounts of glycogen are found in the liver and the kidneys.

The clinical and biochemical aspects of the disease have been studied by di Sant'Agnese and associates.[9] Symptoms have never been observed in the newborn, although they almost always appear before the age of 6 months. The high incidence of consanguinity and of positive family histories in this condition is in sharp contrast to the other diseases under consideration. Sudden death is common, and therapy to date has been unavailing.

Clinical and pathological observations on our three patients are in essential agreement with the data in the literature. We had the unique opportunity in one case to observe the development of the disease from birth to death. Physical examination, electrocardiograms, and chest films revealed no abnormalities at birth and at 2 weeks of age. Cardiomegaly and electrocardiographic changes appeared at the age of 3 weeks, and fatal ventricular fibrillation occurred one week later. At no time did the patient show evidence of congestive heart failure.

From our data and a review of the literature, the following features may help in distinguishing glycogen-storage disease of the heart from the other types of primary myocardial disease: (1) age at onset of symptoms between 2 and 6 months, never beyond 18 months; (2) history of a similar disorder in a sibling; (3) absence of congestive failure in the presence of marked cardiomegaly, and (4) a skeletal muscle biopsy showing a high glycogen content.

Aberrant Left Coronary Artery.—Approximately 30 cases of an aberrant left coronary artery arising from the pulmonary artery have been reported since the

7. Pompe, J. C.: Hypertrophie idiopathique du cœur, Ann. anat. path. **10**:*23*, 1933. Bischoff, G.: Zum klinischen Bild der Glykogen-Speicherungskrankheit (Glykogenose), Ztschr. Kinderh. **52**:*722*, 1932. Putschar, W.: Über angeborene Glykogenspeicherkrankheit des Herzens —"Thesaurismosis glycogenica" (v. Gierke), Beitr. path. Anat. **90**:*222*, 1932.

8. (*a*) di Sant'Agnese, P. A.; Andersen, D. H., and Mason, H. H.: Glycogen Storage Disease of the Heart: II. Critical Review of the Literature, Pediatrics **6**:607, 1950. (*b*) di Sant'Agnese, P. A.; Andersen, D. H.; Mason, H. H., and Bauman, W. A.: Glycogen Storage Disease of the Heart: I. Report of 2 Cases in Siblings with Chemical and Pathologic Studies, ibid. **6**:402, 1950. (*c*) Landing, B. H.; Villee, C., and Nadas, A. S.: Cardiac Glycogen Storage Disease: A Study of 3 Siblings, Proceedings of the New England Cardiovascular Society, Boston, Massachusetts Heart Association, 195-1952. p. 42.

9. Footnote 8 *a* and *b*.

entity was first described, by Abrikosoff,[10] in 1911. At autopsy the patients in these cases show marked cardiac hypertrophy and dilatation with left ventricular myocardial degeneration. Gasul and Loeffler [11] believe that the low pulmonary arterial pressure, rather than the low oxygen content of the coronary arterial blood, causes the myocardial anoxia.

Many of the infants with this disease, as in the case reported here, show episodes of colicky pain with tachycardia, tachypnea, cyanosis, and profuse perspiration.[12] Death commonly occurs during such an episode. These "attacks" are especially likely to accompany feedings, and the picture presented by such a patient suggested to Bland, White, and Garland [5] that the infant was suffering an attack of anginal pain. Frequently, although not invariably, these infants are asymptomatic between "attacks." Some infants with this disease, however, do not give a history of episodal symptoms, and congestive failure is usually absent. A chest film may show an aneurysmal bulging in the region of the left ventricle due to thinning of the myocardium in this area.

In summary, the following features are suggestive of an aberrant left coronary artery: (1) onset of symptoms between 2 and 6 months of age; (2) tachycardia, respiratory distress, cyanosis, and profuse perspiration occurring in "attacks," especially with feedings; frequently the infants are without symptoms between "attacks"; (3) absence of congestive failure, and (4) x-ray evidence of a bulging prominence in the region of the left ventricle.

Medial Necrosis of the Coronary Arteries.—Although an incompletely documented report of a case of this condition was made by Durante [13] in 1899, the first case described in detail was that of Bryant and White [14] in 1901. Since that time, fewer than 30 additional instances of this disease have appeared in the literature.

Pathologically, this is a generalized vascular disease with medial necrosis and intimal proliferation eventually leading to arterial occlusion. The pulmonary and renal vessels, as well as the coronaries, are often affected. The cause of the disease is unknown.

There are few clinical data on the reported cases,[15] and little specific information is available except for the frequent presence of evidence of extracardiac, mainly renal, disease. Death usually occurs before the age of 3 months, and congestive failure is rare. Associated congenital anomalies are not uncommon. The patient

10. Abrikosoff, A.: Aneurysma des linken Herzventrikels mit abnormer Abgangsstelle der linken Koronararterie von der Pulmonalis bei einem fünfmonatlichen Kinde, Arch. path. Anat. **203**:413, 1911.

11. Gasul, B. M., and Loeffler, E.: Anomalous Origin of the Left Coronary Artery from the Pulmonary Artery (Bland-White-Garland Syndrome): Report of 4 Cases, Pediatrics **4**:498, 1949.

12. Kaunitz, P. E.: Origin of Left Coronary Artery from Pulmonary Artery: Review of Literature and Report of 2 Cases, Am. Heart J. **33**:182, 1947.

13. Durante, G.: Athérome congénital de l'aorte et de l'artère pulmonaire, Bull. et mém. Soc. anat. Paris **74**:97, 1899.

14. Bryant, J. H., and White, W. H.: A Case of Calcification of the Arteries and Obliterative Endarteritis, Associated with Hydronephrosis, in a Child Aged 6 months, Guy's Hosp. Rep. **55**:17, 1901.

15. Stryker, W. A.: Arterial Calcification in Infancy with Special Reference to the Coronary Arteries, Am. J. Path. **22**:1007, 1946. Field, M. H.: Medial Calcification of Arteries of Infants, Arch. Path. **42**:607, 1946.

in one of the two cases reported here died within 24 hours after admission; the other was subject to attacks of cyanosis, vomiting, and respiratory distress for four weeks before such an attack led to death.

In summary, medial necrosis of the coronary arteries should be suspected when a patient with evidence of myocardial disease shows the following features: (1) age at onset of symptoms of less than 3 months; (2) evidence of a systemic disease, especially involving the kidneys; (3) associated congenital anomalies, and (4) absence of congestive failure.

Idiopathic Myocarditis.—Idiopathic myocarditis in an adult was first described by Fiedler [16] in 1900. The first instance of this condition in a child was reported in 1901.[17] Since that time, reports of the disease in the pediatric age group have appeared with increasing frequency until at present approximately 100 cases have been recorded. Comprehensive reviews of the subject include those of Saphir, Wile, and Reingold [18] and Tedeschi and Stevenson.[19]

The origin of this disease is unclear. Infectious agents, sensitivity, drugs, vitamin deficiencies, etc., have been suggested as causative factors; but there is no proof that any of these is significant.[20] A futile search for a causative agent in the cases reported in this study included an investigation of drug sensitivity and allergy. Various immunological tests (Schick test, Trichinella skin test, febrile agglutinins, and, when possible, virus antibody titers) were consistently negative.

Idiopathic myocarditis affects older infants and children with greater frequency than babies under 6 months of age. The clinical picture is dominated by the signs and symptoms of right-sided congestive failure. Gallop rhythm or sounds of poor quality occur in a high proportion of cases.

From experience with previous cases and a consideration of possible pathological processes involved, it is felt that the 19 living patients in this report most likely represent instances of idiopathic myocarditis. If we are correct in this assumption, the prognosis of this disease is a great deal more favorable than is generally assumed. Lind and Hultquist,[21] on the basis of their experience with one infant, likewise expressed the opinion that the prognosis need not necessarily be hopeless in this disease.

The excellent response of these patients to the administration of digitalis has not been emphasized sufficiently in the past. We feel that failure of a patient to respond to this medication is evidence against the presence of idiopathic myocarditis.

We agree with the opinion of Weber, Baldwin, and Hall [22] that the diagnosis of idiopathic myocarditis cannot be made with certainty on clinical grounds.

16. Fiedler, A.: Über akute interstitielle Myokarditis, Zentralbl. inn. Med. **21**:212, 1900.

17. Zuppinger, H.: Über Herztod bei anscheinend bedeutungslosen oberflächl. Geschwürsprozessen, Wien. klin. Wchnschr. **14**:799, 1901.

18. Saphir, O.; Wile, S. A., and Reingold, I. M.: Myocarditis in Children, Am. J. Dis. Child. **67**:294, 1944.

19. Tedeschi, C. G., and Stevenson, T. D., Jr.: Interstitial Myocarditis in Children, New England J. Med. **244**:352, 1951.

20. Saphir, O.: Isolated Myocarditis, Am. Heart J. **24**:167, 1942.

21. Lind, J., and Hulquist, G. T.: Isolated Myocarditis in Newborn and Young Infants, Am. Heart J. **38**:123, 1949.

22. Weber, M. W.; Baldwin, J. S., and Hall, J. W.: Acute Isolated Myocarditis: Review of the Literature and a Report of a Case in a 10 Year Old Child, Pediatrics **3**:829, 1949.

Nevertheless, such a diagnosis must be strongly entertained if a patient shows, in addition to the characteristics of the primary myocardial disease group, the following features: (1) age at onset of symptoms beyond 6 months (the likelihood of idiopathic myocarditis increases markedly with an age at onset beyond one year); (2) presence of abnormal heart sounds; (3) evidence of congestive cardiac failure, and (4) good response of congestive failure to digitalis therapy.

Subendocardial Sclerosis.—According to Rauchfuss,[23] subendocardial sclerosis was first described by Kreysig in 1816. Since that time, well over 100 case reports have appeared in the literature. Among the excellent reviews of the subject are those of Craig,[3b] Gross,[24] Cosgrove and Kaump,[25] Prior and Wyatt,[26] Collier and Rosahn,[27] Edmonds and Seelye,[28] and Blumberg and Lyon.[29]

At autopsy, patients with this disease show myocardial hypertrophy and marked endocardial and subendocardial fibroelastic proliferation. Patches of necrosis and degeneration near this fibroelastic reaction are commonly present. Inflammatory infiltrates are conspicuously absent.

The origin of this condition is unknown. For many years, intrauterine infection was thought to be the causative factor.[30] More recently, the disease frequently has been considered to be the result of a developmental error, although cases of adults with very similar pathological changes have been reported.[31] The fact that all of the entities previously discussed, except glycogen storage disease of the heart, frequently show marked endocardial thickening suggests that subendocardial sclerosis may not be primary in itself but rather represents a response of these tissues to some basic stimulus such as anoxia.

Although many cases of valvular involvement in subendocardial sclerosis have been reported, the process did not affect the valves in the cases reported in this study. All patients with valvular involvement in cases reviewed from our pathology files had demonstrated significant murmurs and consequently did not fulfill one of the basic criteria of the clinical syndrome under consideration.

The clinical picture presented by these patients was indistinguishable from that seen in cases of idiopathic myocarditis. Although the babies in this study with

23. Rauchfuss, C.: Die angeborenen Entwicklungsfehler und die Fatalkrankheiten des Herzens und der grossen Gefässe, in Gerhardt, C.: Handbuch der Kinderkrankheiten, Tübingen, Germany, H. Laupp, 1878, vol. 4, Pt. 1, p. 12.

24. Gross, P.: Concept of Fetal Endocarditis: A General Review with Report of an Illustrative Case, Arch. Path. **31**:163, 1941.

25. Cosgrove, G. E., Jr., and Kaump, D. H.: Endocardial Sclerosis in Infants and Children, Am. J. Clin. Path. **16**:322, 1946.

26. Prior, J. T., and Wyatt, T. C.: Endocardial Fibro-Elastosis: Study of 8 Cases, Am. J. Path. **26**:969, 1950.

27. Collier, F. C., and Rosahn, P. D.: Endocardial Fibroelastosis: Report of 2 Cases, Pediatrics **7**:175, 1951.

28. Edmonds, H. W., and Seelye, W. B.: Endocardial Sclerosis: Review of Changing Concepts with Report of 6 Cases, Pediatrics **7**:651, 1951.

29. Blumberg, R. W., and Lyon, R. A.: Endocardial Sclerosis, A. M. A. Am. J. Dis. Child. **84**:291, 1952.

30. Farber, S., and Hubbard, J.: Fetal Endomyocarditis: Intrauterine Infection as the Cause of Congenital Cardiac Anomalies, Am. J. M. Sc. **186**:705, 1933.

31. Gray, I. R.: Endocardial Fibrosis, Brit. Heart J. **13**:387, 1951. Levy, R. L., and Von Glahn, W. C.: Cardiac Hypertrophy of Unknown Cause: A Study of the Clinical and Pathologic Features in 10 Adults, Am. Heart J. **28**:714, 1944.

subendocardial sclerosis first had symptoms at a younger age than the patients with idiopathic myocarditis, others [32] have seen instances of subendocardial sclerosis throughout the pediatric age range. Since we are unable to make a differential diagnosis between subendocardial sclerosis and idiopathic myocarditis, it is possible that some of our living patients suffer from this disease rather than idiopathic myocarditis.

CONCLUSIONS

The above data indicate that five pathologically distinct entities may present an almost identical clinical syndrome. Regardless of cause, patients with this clinical picture consistently showed marked generalized cardiomegaly by x-ray, and in the vast majority, left ventricular hypertrophy and T-wave changes of myocardial damage were found in their electrocardiograms.

These cases may be divided into two subdivisions. One group includes glycogen-storage disease of the heart, aberrant left coronary artery, and medial necrosis of

TABLE 5.—*Clinical Aids in the Etiological Differentiation of Primary Myocardial Disease*

Disease	Age at Onset	Family History	Congestive Failure	Remarks
Glycogen-storage disease of the heart	2-6 mo.	Often positive	Absent	Skeletal muscle biopsy may show high glycogen content
Aberrant left coronary artery	2-6 mo.	Negative	Absent	Symptoms often occur in "attacks"; chest x-ray may show bulge in region of left ventricle
Necrosis of the coronary arteries	Less than 3 mo.	Negative	Absent	May have associated congenital anomalies; often evidence of systemic disease, especially with renal involvement
Idiopathic myocarditis	Throughout pediatric age range	Negative	Present	Gallop rhythm common; congestive failure often responds well to digitalis
Subendocardial sclerosis	Throughout pediatric age range	Negative	Present	Cannot be differentiated clinically from idiopathic myocarditis

the coronary arteries. These are rare conditions which are usually seen in infants under 6 months of age and in which congestive heart failure is uncommon. Idiopathic myocarditis and subendocardial sclerosis constitute the second, and much larger, group. Here the age at onset of symptoms is frequently beyond 6 months, and congestive failure is usually present and often severe. The first group is not amenable to treatment; the second frequently responds to digitalization. Such a division is admittedly not sharp, and there is considerable overlap from one group to the other.

The presence of a positive family history and a skeletal muscle biopsy with high glycogen content (beyond 1.5 mg. per 100 gm.) may be very valuable in detecting patients with glycogen storage disease of the heart. The appearance of symptoms in "attacks" and the demonstration by chest film of a bulging prominence in the region of the left ventricle will occasionally permit the diagnosis of an aberrant left coronary artery. Onset of symptoms before 3 months of age, associated congenital anomalies, and evidence of a systemic disease suggest the presence of medial

32. Glynn, L. E., and Reinhold, J. D. L.: The Relationship of Idiopathic Cardiac Hypertrophy to Foetal Endocarditis, Arch. Dis. Childhood **25**:170, 1950. Sano, M. E., and Anderson, N. A.: Elastic Tissue Hyperplasia of the Endocardium, Arch. Path. **33**:533, 1942.

necrosis of the coronary arteries. Idiopathic myocarditis and subendocardial sclerosis without valvular involvement are indistinguishable by clinical means. No significant sex predominance is seen in any of these diseases.

The clinical features that are helpful in making a differential diagnosis are tabulated in Table 5.

In attempting to establish criteria for the diagnosis of these five diseases in living patients, we had to use data obtained from autopsied patients. It is realized that such a method contains the basic fallacy of applying data obtained from a group suffering from a fatal disease to another group who survived. This approach, however, was the only one available for study of this important problem, and it is felt that the resulting conclusions are valid and should be of considerable assistance to the clinician.

SUMMARY

Forty-five infants and children with a syndrome of myocardial disease characterized by cardiomegaly, absence of significant murmurs, electrocardiographic abnormalities, and normal blood pressure have been studied.

The following pathological processes were found in 26 of these patients who came to autopsy: (1) glycogen-storage disease of the heart (3 patients); (2) aberrant left coronary artery (1 patient); (3) medial necrosis of the coronary arteries (2 patients); (4) idiopathic myocarditis (10 patients), and (5) subendocardial sclerosis (10 patients).

An attempt has been made to establish clinical criteria for the differential diagnosis of these five separate diseases in living patients.

All of these patients showed marked generalized cardiomegaly by x-ray, and the vast majority demonstrated left ventricular hypertrophy and T-wave changes of myocardial damage by electrocardiography.

The value of digitalis in the treatment of some of these patients is stressed.

300 Longwood Ave. (Dr. Nadas).

PULMONARY EDEMA PRODUCED BY INTRATRACHEAL INJECTION OF MILK, FEEDING MIXTURES, AND SUGARS

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PITTSBURGH

DURING the course of a study of experimental milk-aspiration pneumonia produced in rabbits by intratracheal injection of human milk, cow's milk, and feeding mixtures,[1] it was found that injection of 10 to 20 cc. of the feeding mixtures often caused fatal pulmonary edema within a few minutes. Injections of similar amounts of human or cow's milk rarely produced this effect.

Evaluation of the contents of the feeding mixture suggested that the difference in the effects of the injection of the feeding mixture as compared to injection of

TABLE 1.—*Pulmonary Edema and Death from Injections of Milk and Feeding Mixtures*

Material	No. Rabbits	Pulmonary Edema	Death from Pulmonary Edema
Feeding mixture (1 injection, 3 cc.)	7	..	..
Human milk (1 injection, 3 cc.)	2	..	..
Cow's milk (1 injection, 3 cc.)	2	..	..
Feeding mixture (3 or 4 cc. on 3 consecutive days)	6	..	..
Human milk (3 or 4 cc. on 3 consecutive days)	4	..	..
Cow's milk (3 or 4 cc. on 3 consecutive days)	5	..	..
Feeding mixture (5-cc. amounts 17 times in 22 days)	1	..	..
Feeding mixture (1 injection 10-20 cc.)	14	9	9
Human milk (1 injection 15 or 20 cc.)	2	..	..
Cow's milk (1 injection 10-20 cc.)	4	1	1
Cow's milk (repeated injections 10-20 cc. at 15-min. intervals up to 70 cc.)	7	2	2

unfortified human milk or cow's milk was due to the larger amount of carbohydrate in the feeding mixture. This led to a more detailed study of the production of acute pulmonary edema by intratracheal injection of feeding mixtures, human milk, cow's milk, and solutions of the carbohydrates present in milk and the common feeding mixtures.

METHODS

Intratracheal injections of feeding mixture (2 oz. [59.2 ml.] evaporated milk, 5 oz. [147.9 ml.] distilled water, and 1 tablespoon [15 ml.] Dextri-Maltose No. 1), cow's milk, human milk, and varying concentrations of the four principal carbohydrates used in feeding mixtures (lactose, dextrose, maltose, and dextrin) were given to 105 rabbits. Most of the injections were given under ether anesthesia, but a few were given without anesthesia. The amounts of the various

From the Department of Pathology and the John C. Oliver Memorial Research Foundation, St. Margaret Memorial Hospital, and the Department of Pathology, University of Pittsburgh School of Medicine.

1. Moran, T. J.: Milk-Aspiration Pneumonia in Human and Animal Subjects, A. M. A. Arch. Path. **55**:286, 1953.

TABLE 2.—*Pulmonary Edema and Death from Injection of Twenty- and Ten-Cubic-Centimeter Amounts of Carbohydrates*

Material	20 Cc.	No. Rabbits	P. E.	Died	10 Cc.	No. Rabbits	P. E.	Died
Dextrose	50%	1	+	+	50%	2	+	+
Dextrose	25%	1	+	+	25%	1	+	+
Dextrose	12.5%	1	+	+	12.5%	1	—	—
Dextrose	6.25%	1	—	—	6.25%	1	—	—
Maltose	50%	2	+	+	50%	1	+	+
Maltose	25%	1	+	+	25%	1	—	—
Maltose	12.5%	1	+	—	12.5%	1	—	—
Maltose	6.25%	1	—	—	6.25%	1	—	—
Lactose	50%	2	+	+	50%	1	+	+
Lactose	25%	1	+	+	25%	1	—	—
Lactose	12.5%	1	+	—	12.5%	1	—	—
Lactose	6.25%	1	—	—	6.25%	1	—	—
Dextrin	50%	1	+	+	50%	1	—	—
Dextrin	25%	1	+	+	25%	1	—	—
Dextrin	12.5%	1	—	—	12.5%	1	—	—
Dextrin	6.25%	1	—	—	6.25%	1	—	—

TABLE 3.—*Pulmonary Edema and Death from Other Mixtures*

Material	No. Rabbits	Pulmonary Edema	Death from Pulmonary Edema
Dextrose supersaturated sol. (10 cc.)	1	+	+
Dextrose 50% (7.5 cc.)	1	+	+
Dextrose 50% (8 cc.)	1	+	—
Dextrose 50% (5 cc.)	1	+	—
Dextrose and maltose 25% (20 cc.)	1	+	+
Dextrose 25% (5 cc.)	2	—	—
Lactose 25% (5 cc.)	2	—	—
Dextri-maltose 15 gm. in 100 cc. H_2O (30 cc.)	2	+	+
Dextri-maltose 15 gm. in 100 cc. H_2O (45 cc.)	1	+	—
Dextri-maltose 15 gm. in 100 cc. H_2O (15 cc.)	1	—	—
Dextri-maltose 7.5 gm. + lactose 5 gm. in 100 cc. H_2O (20 cc.)	2	+	+
Dextri-maltose 7.5 gm. + lactose 5 gm. in 100 cc. H_2O (10 cc.)	1	—	—

TABLE 4.—*Total Protein and Sugar Determinations of Edema Fluid in Fourteen Rabbits with Pulmonary Edema*

Wt., Lb.	Material Injected, Amount (Cc.)	Death, Min.	Wt. Lungs, Gm.	Edema Fluid, Cc.	Protein Content Edema Fluid, Mg./100 Cc.	Sugar Content Edema Fluid, Gm./100 Cc.
8.8	50% dextrose 8	9	30	15	364	28 dextrose
10	50% dextrose 5, 1st day 4, 2d day	15 (after 2d injection)	34	3.1	1,180	8 dextrose
8	50% dextrose 5	30 (killed by air)	35	20	727	18 dextrose
4.2	Cow's milk 40	35	17	8.5	2,944	3.5 lactose
4.5	Cow's milk 70	180 (killed by air)	21	3.0	3,210	2.4 lactose
5.5	18.5% dextrose 10	3	38	35	198	14.8 dextrose
5.5	25% dextrose 20	3	30	35	79	9.6 dextrose
5.5	12.5% dextrose 20	4	22	13	None	5.2 dextrose
5.5	25% lactose 20	0.5	25	18	None	12 lactose
5.5	12.5% lactose 20	4 days (killed by air)	13.5	10	79	1.7 lactose
4.8	25% maltose 20	4.5	22	11	79	6.2 maltose
4.5	12.5% maltose 20	(Still alive)	..	7	506	6.1 maltose
6.1	25% dextrose 10	14	28	16	None	5.6 dextrose
4	50% lactose 10	10.5	26	15.5	None	6.2 lactose

materials injected, the occurrence of pulmonary edema, and the deaths from pulmonary edema are shown in Tables 1, 2, and 3. Pulmonary edema was not diagnosed unless frothy edema fluid appeared at the nose and mouth.

The edema fluid from 14 of the rabbits who developed pulmonary edema was collected and measured, and total protein and sugar determinations of the fluid were made. The total protein determinations were done by the macro-Kjeldahl method, and the sugar determinations were made by the Folin-Wu technique. These results as well as the weights of the animals, the weights of the lungs, and the time of death are shown in Table 4.

Four guinea pigs were also used in the experiment, to see if pulmonary edema could be produced in guinea pigs by intratracheal injection of milk, feeding mixture, or carbohydrate solutions. Intratracheal injections of hypertonic sodium chloride were given to three rabbits. Other rabbits were given intratracheal injections of distilled water in amounts up to 100 cc. When injections greater than the capacity of the rabbit's lungs and trachea were used, they were given in divided doses of 20 cc. every 10 or 15 minutes.

RESULTS

In many animals given injections acute pulmonary edema developed, which in most instances was fatal. The pulmonary edema usually developed in 30 seconds to 5 minutes, but it occasionally took as long as 30 minutes to occur. Death occurred in from 2 to 35 minutes after the injections. The animals showed increased respiratory and pulse rates, convulsive jerking of the head or the entire body, and spasticity, followed by a sudden gush of frothy white, pink, or occasionally bloody fluid. This fluid was often ejected violently, sometimes spraying an area several feet in diameter. The froth continued to appear for 30 seconds to 3 or 4 minutes. At the end of this time the animal usually died.

Pulmonary edema, as manifested by the production of frothy edema fluid at the nose and mouth, did not develop in any of 27 rabbits when 5 cc. or less of human milk, cow's milk, or feeding mixture was injected intratracheally either on one occasion or on consecutive days (Table 1).

Single intratracheal injections of 10, 15, or 20 cc. of feeding mixture produced fatal pulmonary edema in 9 of 14 rabbits. Single injections of 20 cc. of cow's milk produced fatal pulmonary edema in one of four rabbits. Repeated injections of 20 or 10 cc. of cow's milk approximately every 15 minutes up to a total of 70 cc. produced fatal pulmonary edema in two of seven rabbits. Injection of 15 or 20 cc. of human milk did not cause pulmonary edema in either of two rabbits. The amounts of milk or feeding mixture, the number of animals used, and the results are shown in Table 1.

Intratracheal injections of 20 cc. of 50% or 25% dextrose, maltose, lactose, or dextrin produced fatal pulmonary edema in all of 10 rabbits. Injection of 20 cc. of 12.5% solutions of dextrose, maltose, or lactose produced acute pulmonary edema in each of three animals, but the animals given the maltose and lactose solutions recovered. In one animal pulmonary edema did not develop after injection of 20 cc. of 12.5% dextrin. Injection of 20-cc. amounts of 6.25% solution of dextrose, maltose, lactose, or dextrin, did not cause pulmonary edema in any of the four rabbits.

When 10-cc. amounts of carbohydrate solution were injected, the 50% solution of dextrose, maltose, or lactose caused fatal pulmonary edema in all of four animals. Injection of 25% dextrose caused fatal pulmonary edema in one rabbit, but 25% maltose, lactose, or dextrin did not cause pulmonary edema. Injection of 10-cc. amounts of 12.5% or 6.25% solutions did not cause pulmonary edema in any of eight rabbits. The strength and amount of the carbohydrate solutions injected and the results are shown in Table 2.

The amounts of a variety of other materials which were injected and the results of the injection are shown in Table 3. Amounts of 5 cc., 7.5 cc., and 8 cc. of 50% dextrose produced acute pulmonary edema in each of three animals, but two of the animals recovered. Injection of 5-cc. amounts of 25% dextrose or 25% lactose did not cause pulmonary edema in any of four rabbits. Thirty- and 45-cc. amounts of Dextri-Maltose No. 1 solution without milk (15 gm. in 100 cc. of distilled water) produced pulmonary edema in each of three rabbits. One rabbit recovered. Injection of 20-cc. amounts of Dextri-Maltose No. 1 solution to which lactose was added but no milk (7.5 gm. Dextri-Maltose No. 1 and 5 gm. of lactose in 100 cc. of distilled water) caused fatal pulmonary edema in two rabbits. Ten cubic centimeters of this same solution injected into one rabbit did not cause pulmonary edema.

Total protein determinations of the edema fluid from most of the animals showed varying amounts of protein (Table 4). In general the highest protein determinations were found in the animals which lived longest. The lowered sugar content of the edema fluid as compared to the injected solutions was caused by the outpouring of the protein-containing fluids from the alveolar capillaries.

Four guinea pigs were given intratracheal injections of one each of the following solutions: 3 cc. of 50% dextrose, 3 cc. of 50% lactose, 4 cc. of feeding mixture, and 5 cc. of cow's milk. Fatal pulmonary edema developed in all of them.

In three animals injected with hypertonic sodium chloride solution fatal pulmonary edema developed. One animal received 20 cc. of 36% sodium chloride, one animal 7.5 cc. of 18%, and one animal 20 cc. of 8%. In rabbits injected with distilled water in amounts up to 100 cc. pulmonary edema did not develop. The distilled water was given in repeated doses of 20 cc. every 10 or 15 minutes.

COMMENT

In this experiment acute pulmonary edema was rarely caused by intratracheal injection of cow's milk unless carbohydrates had been added to the milk. Addition of enough sugar to make a carbohydrate solution of 12.5% (including the approximately 5.0% lactose normally present in the milk) usually caused fatal pulmonary edema. Injection of 20 cc. amounts of 12.5% or stronger solutions of dextrose, lactose, or maltose also produced pulmonary edema, while the same amount of 6.25% solutions did not. Pulmonary edema was produced by injection of 10-cc. amounts of these carbohydrates only when 25 or 50% solutions were used. The results indicate that the production of the pulmonary edema is due to difference in osmotic pressure because of the hypertonicity of the solution. The volume and the concentration of the injected material are both important.

Melli and Pisa (1931)[2] produced acute pulmonary edema in rabbits by intratracheal injection of concentrated glucose solutions, and they concluded that the hypertonicity of the solution caused the edema. They believed that the mechanism of death was asphyxia caused by the filling of all available air space by the edema fluid. This interpretation of the mechanism of death is open to question. In our experiment many animals were given quantities of fluid far in excess of the capacity of the lungs. The fluid capacity of the trachea and lungs of the rabbits in this series

2. Melli, G., and Pisa, M.: Ricerche sperimentali sull'edema polmonare acuto, Minerva med. **2**:419, 1931.

varied from 10 to 20 cc. depending on the size of the animal. As long as the air passages were not mechanically obstructed, the air continued to get into the lungs, and the excess fluid bubbled out of the nose and mouth. Production of the frothy pulmonary edema fluid in this series was usually preceded by or accompanied by violent convulsive movements of the head or entire body and spasticity of the body, the appearance suggesting a central nervous system irritation.

Clinical application of the results of this experiment is suggested for a variety of patient feeding problems especially in infancy and old age. Administration of milk to which carbohydrate has been added or administration of strong carbohydrate solution alone would seem hazardous in infants with feeding problems, especially those receiving feeding by gavage. Some of the unexplained sudden deaths in infants during or following feeding may be due to acute pulmonary edema not diagnosed clinically and at autopsy regarded as a finding secondary to an undiscovered primary condition. Some of the instances of clinically diagnosed acute or subacute pulmonary edema in infants, not previously explained, may also be due to aspiration of milk or feeding mixtures, especially those with a high-carbohydrate content.

In treatment of infants with feeding problems, when milk is not used because of the danger of aspiration pneumonia, the physician should also avoid the use of strong carbohydrate solutions. The results reported suggest that a carbohydrate concentration comparable to that in cow's milk (approximately 5.0%) would rarely cause acute pulmonary edema even when aspirated in relatively large quantities, but that increasing carbohydrate concentrations rapidly become dangerous. For the first few days of life, at least, only water should be given by mouth to infants with feeding problems, and necessary carbohydrates should be given by other routes.

The production of acute pulmonary edema by milk or carbohydrate solution is probably not so important in adults as in infants. However, there are many references in the psychiatric literature to acute pulmonary edema as a complication of insulin therapy in mental patients.[3] These reactions have usually been ascribed to use of epinephrine but many of the patients in reported cases had received sugar solutions by mouth, and some of the reactions may have been due to gavage feeding of strong dextrose solution where the material was accidentally injected directly into the trachea or where it was aspirated after vomiting. Use of strong carbohydrate solutions by mouth may also be dangerous in other adult patients with impaired reflexes or in patients who are vomiting.

SUMMARY

The production of acute, often fatal, pulmonary edema in rabbits and guinea pigs by intratracheal injection of feeding mixtures or carbohydrate solutions (lactose, dextrose, maltose, and dextrin) has been described. The production of pulmonary edema is quite consistent, and the severity of the reaction can readily be controlled

3. O'Neill, F. J.: Serious Complications of Insulin Shock Therapy, Psychiatric Quart. **12**:455, 1938. Furst, W.: Insulin Shock Treatment: Death Due to Pulmonary Gangrene, Am. J. Psychiat. **97**:380, 1940. Gottesfeld, B. H.: Use of Adrenal Cortex Extract in Cases of Insulin Shock Complicated by Pulmonary Edema: Review of Literature and Report of a Case, Arch. Neurol. & Psychiat. **49**:304, 1943. Gralnick, A.: Pulmonary Edema and Electrocardiographic Findings Resembling Coronary Occlusion in Insulin Treatment: Report of a Case, Psychiatric Quart. **18**:650, 1944.

by the concentration of the solution and the volume of material injected. Feeding mixtures containing carbohydrates in addition to the lactose normally present become more dangerous as the carbohydrate content is increased. The production of the pulmonary edema probably results from the difference in osmotic pressure between the fluid in the alveolar capillaries and the injected hypertonic solution in the alveolar spaces.

The findings reported suggest that some of the sudden deaths occurring in infants during or shortly after feeding may be due to pulmonary edema caused by aspiration of feeding mixture into the lungs, and that some instances of acute pulmonary edema in infants, not explained by other findings, may be due to aspiration of feeding mixture to which carbohydrates have been added. Human milk and cow's milk to which carbohydrate has not been added do not involve nearly so great a risk as the feeding mixtures. Infants with feeding problems should be given only water by mouth until feeding habits are established. Other required materials may be given by other routes.

The findings reported also suggest the potential danger of administration of feeding mixtures or carbohydrate solutions to adults with feeding problems, especially those requiring gavage feedings. Strong sugar solutions such as those used in gavage feeding in insulin-shock therapy may be especially dangerous. Use of strong sugar solution by mouth may also be hazardous in patients who are vomiting because of the possibility of aspiration of the vomitus.

Proper feeding methods are important in preventing acute pulmonary edema as well as in preventing pneumonia caused by aspiration of milk.

Case Reports

COCCIDIOIDOMYCOSIS IN INFANTS

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IT HAS BEEN emphasized by Kunstadter and Pendergrass [1] that coccidiodomycosis has not received the attention it deserves from a pediatric standpoint. The purpose of this article is to present the case of the youngest autopsied patient with coccidioidomycosis.

REPORT OF A CASE

K. F. B., a 3-week-old white girl, was admitted to Kern General Hospital on July 25, 1952, with a history of irritability, anorexia, and high fever for two days. She had been seen by a local physician, who did a lumbar puncture, obtaining cloudy fluid. She was referred to the Kern General Hospital contagious service for diagnosis and treatment.

Summary of Laboratory Findings on Examination of Spinal Fluid

Date	Cells	Differential Count		Sugar, Mg./100 Cc.	Protein, Mg./100 Cc.	Culture	Subdural
		Neutrophiles	Lymphocytes				
7/25/52	700	80%	20%	15	195	Negative for bacteria	.....
7/31/52	23	9%	91%	49	64	Negative for bacteria	.....
8/ 3/52	5	40%	60%	45	53	Negative for bacteria	Negative
8/16/52	46	16%	84%	43	46	Negative for acid-fast and Coccidioides immitis	Negative
8/25/52	98	72%	28%	34	53	Positive for C. immitis	.....

Review of Systems.—There was no history of convulsions, tremors, or twitchings and no vomiting or diarrhea.

Past History.—The baby was born by spontaneous delivery and weighed 5 lb. (2,270 gm.). She was taken home on the third day. There was no previous illness.

Family History.—The mother and father were in good health. There was no history of tuberculosis, diabetes, syphilis, or any other diseases. They had been living in the San Joaquin Valley for 12 years. The father works as a foreman on one of the fruit farms.

Physical Examination.—Physical examination revealed an acutely ill infant with a temperature of 104 F. (rectal) and a pulse rate of 132. The skin was a little pale. The throat was mildly injected. There was questionable nuchal rigidity. The fontanel was full but not tense. No other abnormalities were noted.

Laboratory Findings.—Spinal fluid findings are listed in the table. Findings on urinalyses, including cultures for bacteria and Coccidioides immitis, were consistently negative. The hemoglobin was 12.3 gm. per 100 cc. on admission and dropped to 8.2 gm. within one week, returning to 11.1 gm. following transfusions. The red blood cell count ranged around 3,500,000. The white blood cell count varied from 7,100 to 19,800, with a relative normal differential count.

From the Pediatric Department, Kern General Hospital (Dr. McKey).

1. Kunstadter, R. H., and Pendergrass, R. C.: Primary Coccidioidomycosis: A Possible Pediatric Problem, J. A. M. A. **127**:624-627 (March 17) 1945.

Blood cultures, bone marrow examinations, and gastric washings failed to show C. immitis. Blood tests for heterophilic antibodies and agglutinations for typhoid, typhus, and brucellosis were negative. The result of serology tests for coccidioidomycosis was 4 + in first dilution of complement fixation, which was thought to be a false positive.

Clinical Course.—Owing to low spinal fluid sugar and high blood cell count, it was felt that this was most likely a case of bacterial meningitis. Administration of streptomycin, penicillin, and triple sulfonamides was started. The temperature was down by lysis on the fifth hospital day. This was considered a good response, so administration of streptomycin was discontinued. A tranfusion of 125 cc. of whole blood was given on Aug. 3, 1952. The temperature began to spike between 100 and 103 F. (all temperatures are rectal). Physical examination showed no significant abnormalities. The child continued to eat well and did not appear in any distress. A chest x-ray on Aug. 8, 1952, revealed patchy infiltration of both lungs, more marked on the left. It was felt that the pneumonia was resistant to penicillin and sulfonamide therapy. These drugs were discontinued and administration of 250 mg. of oxytetracycline (Terramycin) every six hours was started. The temperature continued to spike from 100 to 104 F. The first- and second-strength skin tests for tuberculosis and coccidioidomycosis were negative. On Aug. 14, 1952, an enlarged spleen was noted. Chest x-ray on Aug. 21, 1952 revealed patchy nodular infiltration throughout both lung fields suggestive of miliary tuberculosis or coccidioidomycosis. The baby appeared to be going downhill and refused feedings for the first time. In view of the x-ray findings and the evidence of chronic meningitis with failure to respond to any of the antibiotics, it was felt that tuberculosis or coccidioidomycosis existed. Administration of isonicotinic acid hydrazide (Rimifon), streptomycin, and *p*-aminosalicylic acid was started. Spinal fluid culture done on Aug. 25, 1952, was reported on Sept. 3, 1952, as positive for C. immitis. On Sept. 4, 1952, scattered papules over the trunk were noted. There were enlarged cervical nodes. The spleen remained palpable two to three fingerbreadths below the left costal margin. Skeletal x-ray failed to reveal any involvement. The infant died Sept. 5, 1952, at the age of 2 months. A 36-hour reading of a repeat coccidioidal skin test was negative.

An autopsy was performed which demonstrated disseminated coccidioidomycosis. Grossly, the process involved the lungs, liver, spleen, kidneys, pancreas, adrenals, and possibly the endocardium of the heart. There were small reddish nodules on the mitral valve and several small yellow-white nodules on the tricuspid valve. These nodules appeared to be a granulomatous inflammatory reaction rather than the acute destructive reaction of a bacterial endocarditis. Both lungs were about two to three times their normal weights. There were numerous subpleural granulomatous nodules. On sectioning the lung, it was found that there was very little normal tissue left. It was replaced by chronic inflammatory reaction consisting of nodular areas measuring up to 8 mm. in diameter throughout both lung fields. A smear from these lesions revealed typical C. immitis spherules. The spleen revealed many lesions of similar size. The lesions in the other organs were numerous but smaller in size. Microscopically, the mitral and tricuspid valves were thickened and fibrotic, showing a chronic inflammatory reaction. There were several dilated blood vessels in the valves, but no etiologic agent was seen to explain the valvulitis. In the lungs the greater part of the parenchyma had been replaced by a chronic granulomatous reaction of a both proliferative and destructive nature. Numerous Coccidioides spherules were present, some containing hundreds of endospores, one measuring 260 μ in diameter and many measuring over 150 μ in diameter. The damage to the brain was considerable; however, C. immitis was seen in only one section. Sections of the thyroid, thymus, pancreas, adrenal, kidneys, liver, spleen, and adjacent lymph nodes showed focal areas of caseous necrosis and many coccidioides spherules.

COMMENT

A review of the literature reveals only one case of disseminated coccidioidomycosis in a patient less than one month of age.[2] This was the case of a child with spina ventosa, who was still living four years later. One other patient of less than

2. Cohen, R.: Coccidioidomycosis: Case Studies in Children, Arch. Pediat. **66**:241-265 (June) 1949.

one month of age has been reported as having a positive skin test with no evidence of active coccidioidomycosis at that time.[3]

The possibility of maternal transmission could not be proved. The mother had a positive skin test, but serology could not be done because of lack of cooperation. Cohen and Burnip [3] in two cases and Smale and Birsner [4] in four cases were unable to demonstrate placental transmission.

The rapidity with which the disease disseminated would leave the impression that the newborn infant has very little resistance against the organism. The spinal fluid changes on admission would suggest dissemination at that time.

The negative coccidioidin skin tests are unusual. It might be postulated that the infection was so overwhelming that the infant could not develop any antibodies. It is generally known that the skin test may become negative in the terminal stage. The negative complement fixation test would seem to indicate the same.

The autopsy findings are given in some detail to demonstrate the overwhelming process. The body had been embalmed, yet cultures of the pulmonary tissue readily grew C. immitis. The endocardial lesions are of interest in that only one case could be found.[5] Although no organism could be found, it seems fairly certain that C. immitis was the cause of the endocarditis. The spherules in the lung and the spleen were among the largest ever seen.

CONCLUSIONS AND SUMMARY

A case of disseminated coccidioidomycosis in a 3-week-old infant with death at two months is reported. The disease may be found at any age and appears to be quite fulminating during infancy. Some unusual findings are reported.

3. Cohen, R., and Burnip, R.: Coccidioidin Skin Testing During Pregnancy and in Infants and Children, California Med. **72**:31-33 (Jan.) 1950.

4. Smale, L. E., and Birsner, J. W.: Maternal Deaths from Coccidioidomycosis, J. A. M. A. **140**:1152-1154 (Aug. 6) 1949.

5. Epstein, E.: Prognostic Significance of Cutaneous Lesions in Coccidioidal Granuloma, Arch. Dermat. & Syph. **38**:752-755 (Nov.) 1938.

OSTEOCHONDRITIS DEFORMANS COXAE JUVENILIS

Familial Demonstration

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THE EPIPHYSIS is that portion of bone concerned with its length growth. It develops from a secondary center of ossification and is separated from the main portion of bone by unossified cartilage. This unossified layer is known as the epiphyseal plate and disappears only after growth has ceased. The physiological changes occurring in these regions are, of necessity, associated with marked vascularity to accommodate the increased cellular activity.

The terms, epiphysitis, osteochondritis, and osteochondrosis have been used to signify a derangement of the normal process of bone growth. Although any epiphysis may become disturbed in a process of this type, its particular location may predispose it to changes. Originally, these disturbances were described separately and each named for its discoverer. The literature is therefore confused by individual designation of diseases which should be grouped under one disease entity, namely, osteochondrosis. The latter has been accepted in the standard nomenclature of disease.

Osteochondrosis of the hip, also known as osteochondritis deformans coxae juvenilis, or coxa plana, was described independently by Legg,[1] by Perthes,[2] and by Calvé,[3] hence, it is occasionally known as Legg-Perthes-Calvé disease. It is a disease occurring usually between the ages of 4 and 10 years, involving boys predominantly, being unilateral in 90% of cases, characterized by degenerative changes in the epiphysis of the femoral head, and usually running a self-limited course. The cycle of events from original degeneration to complete replacement varies from 18 months to five years. Howorth[4] describes four definite stages in the cycle: (1) the early stage, lasting several weeks with changes in soft tissues and mild symptoms associated with synovitis; (2) the active stage, lasting a year or two, in which degeneration in the head and softening at the epiphyseal disk occur; (3) the healing stage,

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1. Legg, A. T.: An Obscure Affection of the Hip-Joint, Boston M. & S. J. **162**:202-204, 1910.

2. Perthes, G. C.: Über Arthritis deformans juvenilis, Deutsche Ztschr. Chir. **107**:111-159, 1910.

3. Calvé, J.: Sur une forme particulière de pseudo-coxalgie graffée sur des déformations caractéristiques de l'extrémité supérieure du fémur, Rev. de chir., Paris **30**:54-84, 1910.

4. Howorth, M. B.: Coxa Plana, J. Bone & Joint Surg. **30A**:601-620, 1948.

lasting three to four years, in which dense areas are replaced by normal bone; (4) the residual stage, in which only the deformity, coxa plana, remains. This division into stages is well accepted.

The cause of coxa plana, however, is not known. Doubtless this is the result of the few opportunities for histological studies, since operative intervention is seldom indicated. Trauma is considered as a major factor by some, as an incidental factor by others; certainly the preponderance of boys in any series would strongly suggest its importance. Endocrine disturbances are suggested; there is no definite proof of this relationship, particularly since the majority are unilateral involvements. Infection is suspected because of associated swelling, tenderness, and early signs of sepsis. A combination of the above has been suggested by Mercer.[5] Familial or hereditary relationship has not been demonstrated.

The observation and treatment of patients in three cases of osteochondrosis of the hip in one family prompted this report. No other epiphyseal changes have been found. The family history was negative.

REPORT OF CASES

CASE 1.—D. C., a boy aged 10 years, was seen June 1, 1944, with the complaint of left hip pain of two years' duration. Six months after onset, immobilization in a single-hip spica cast and bed rest for three months failed to relieve him, and treatment was discontinued.

Examination showed legs of equal length, but restricted left hip motion allowing only 15 degrees abduction, no internal rotation beyond neutral, only 15 to 20 degrees of external rotation, and a positive reaction to the Trendelenberg test. X-ray examination on June 1, 1944, demonstrated a healed process on the right femur, a defect in the superior aspect of the head of the left femur, suggesting incompletely healed necrosis, and both heads with mild coxa plana (Fig. 1).

This patient was fitted with a left long-leg ischial-bearing walking caliper brace and crutches. A cooperative and observing parent kept the ischial ring elevated to carry the patient's weight consistently during subsequent growth. Hip pain subsided gradually. Brace support was discontinued Dec. 10, 1944, and crutches were retained to Feb. 1, 1945.

The last examination, on April 1, 1948, demonstrated legs of equal length, free hip range, and a negative reaction to the Trendelenburg test. X-ray examination demonstrated good replacement and contour of both femoral heads with mild coxa plana changes (Fig. 2).

CASE 2.—R. C., a boy aged 8 years, was seen June 1, 1944, with the complaint of pain in the right groin and a right-sided limp of three months' duration. He was treated elsewhere immediately by immobilization in a single-hip spica cast.

Examination showed marked right hip joint restriction in all planes and a marked adduction contracture. X-ray examination demonstrated a dense slightly flattened head of the femur on the right side as in an advanced osteochondrosis (Fig. 3).

Under general anesthesia, the hip joint contracture was corrected by manipulation and a double spica cast applied for three months, incorporating tibial tuberosity pins. The hip was subsequently protected from weight-bearing by a right long-leg ischial-bearing caliper brace and two crutches. This patient was very active and difficult to control; periodically he was seen without brace or crutches. Gradual healing was observed on subsequent x-ray examinations, with final study of May 25, 1951, showing coxa plana, coxa vara, and a short femoral neck (Fig. 4).

The physical findings include ½ in. (1.3 cm.) shortening of the right leg, restricted abduction and extension, 30 degrees flexion contracture, and mild limp. Osteotomy has been suggested.

5. Mercer, W.: Orthopaedic Surgery, Ed. 4, Baltimore, Williams & Wilkins Company, 1950, p. 454.

Fig. 1 (Case 1).—X-ray showing a healed process on the right femur, a defect in the superior aspect of the head of the left femur suggesting incompletely healed necrosis, and both heads with mild coxa plana.

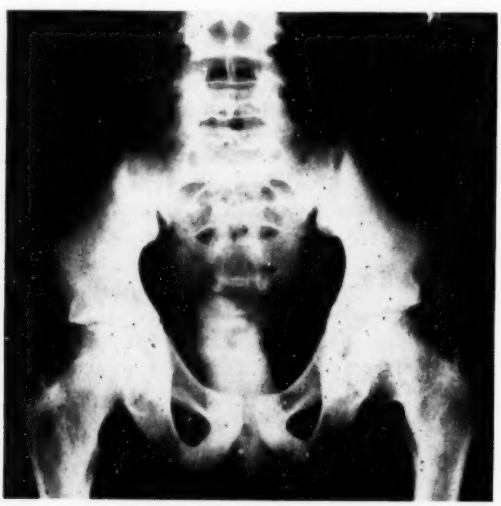

Fig. 2 (Case 1).—X-ray showing good replacement and contour of both femoral heads with mild coxa plana changes.

Fig. 3 (Case 2).—X-ray showing a dense slightly flattened head of the femur on the right side, as in advanced osteochondrosis.

Fig. 4 (Case 2).—X-ray showing coxa plana, coxa vara, and a short femoral neck.

Fig. 5 (Case 3).—X-ray showing slight atrophy of the femoral head and flattening of its superior surface.

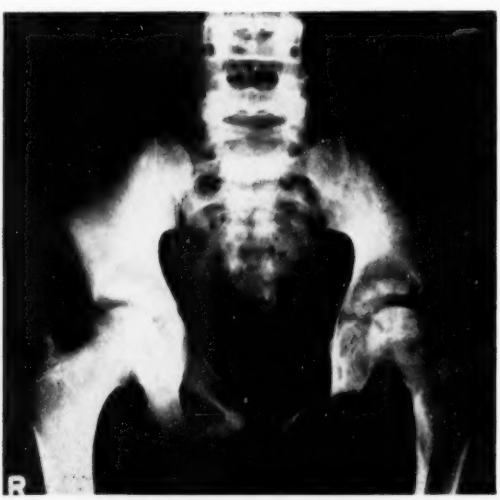

Fig. 6 (Case 3).—X-ray showing necrosis of the greater part of the femoral head, with beginning absorption at both medial and lateral poles.

CASE 3.—T. C., a boy aged 11 years, was seen April 25, 1952, after the parents had noted the child walked with a left-sided limp. A history of a minor fall off a bicycle three months before was elicited.

Examination showed legs of equal length, left hip joint mildly restricted in motion in all planes, particularly in abduction and internal rotation, and a positive Trendelenburg test. X-ray examination demonstrated slight atrophy of the femoral head and flattening of its superior surface (Fig. 5).

The child was immobilized in a double spica cast for four months, then fitted with a long-leg ischial-bearing caliper brace and crutches. X-ray examination on Oct. 3, 1952, demonstrated necrosis of the greater part of the femoral head, with beginning absorption at both medial and lateral poles (Fig. 6). Protection of the joint has been maintained to date.

SUMMARY

The occurrence of osteochondritis deformans coxae juvenilis in three brothers suggests a familial predisposition to the disease. This predisposition may be either an inherent hip weakness or a familial endocrine change. A combination of factors, rather than one factor alone, seems to be necessary in the development of this disability.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

George F. Munns, M.D., President
Regular Meeting, Jan. 20, 1953

Cystic Fibrosis of the Pancreas. DR. B. M. KAGAN.

D. M., a 7-month-old infant, was admitted to the hospital on June 25, 1952. The case is presented because some of the pertinent and significant features of the disease are illustrated and also in order to demonstrate a new metabolic phenomenon found in the research laboratory. The child was sent to the hospital because his physician was having difficulty in establishing a diagnosis. Cystic fibrosis of the pancreas was suspected, but repeated stool examinations were positive for trypsin; one duodenal drainage was also positive for trypsin.

When 2 months of age, the infant had an upper respiratory infection for which he was given oxytetracycline (Terramycin) and aureomycin; he improved. At the age of 5 months, he passed loose foul-smelling stools four or five times daily. The stools often contained undigested food. A bronchiolitis which occurred at this time responded to antibiotic therapy. He was given Panteric (triple-strength pancreatin [Parke, Davis & Company]) granules empirically, but did not gain well.

Family History.—About nine years prior to this child's birth, the mother gave birth to a child who died at the age of 6 months; death was attributed to "virus pneumonia." This infant had developed poorly, although he ate well; he too had had foul-smelling stools. No autopsy was performed. In her next pregnancy the mother had a miscarriage in the second month. Her next pregnancy resulted in a child now 7 years old and normal; there is another normal child, a 4½-year-old girl. The fifth pregnancy resulted in a boy baby who died, when 7 hours old, of a meconium ileus.

The present patient was the result of a sixth pregnancy and one of twins, the twin brother dying of a meconium ileus. Physical examination revealed an undernourished pale infant with a relatively large abdomen, a moderate amount of nasal discharge blocking the nares, and small but not wasted buttocks. X-ray examination of the chest showed no abnormalities.

The duodenal drainage fluid was a light brown fluid of neutral pH. It was negative for trypsin. The viscosity was over 11 minutes; normal has been less than 2 minutes by our method. Four stools on consecutive days were examined for trypsin; two were negative, and two were positive. Stool culture showed Proteus vulgaris. All stools were negative for fat, and most showed a moderate amount of intracellular starch. Results of the stool eosinophile test were negative. The vitamin A palmitate in aqueous dispersion showed 45 units at 0 hour, 249 at 3 hours, 96 at 6 hours and 51 at 24 hours. The vitamin A alcohol showed 24 at 0 hour, 147 at 3 hours, 357 at 6 hours and 27 at 24 hours. The vitamin A percomorph liver oil showed 17 at 0 hour, 39 at 3 hours, 38 at 6 hours and 27 at 24 hours.

The child was started on a regimen which included a low-fat high-protein moderate-starch diet, Viokase (a whole pancreas powder), high intake of aqueous dispersion of vitamins A and D, and vitamins C and B. He was given one dose of oxytetracycline daily in the morning for prophylaxis. At the last written report from his doctor, his growth was normal as compared with a normal channel on the Iowa growth grid. There appears to be an excellent chance that this child will survive his first year and at least a 50% chance that he will survive beyond the age of 5.

Gastric Bleeding in the Newborn. DR. JOSEPH GREENGARD.

This baby was born of a 15-year-old mother. Delivery was uneventful, and the baby was about to be discharged on the third day when blood was noted on the blanket. No abnormal findings were noted on examination, but several episodes of vomiting occurred that day, all of which consisted of bright red blood. The infant was then transferred to Sarah Morris Hospital for Children, and various diagnostic tests were instituted.

The prothrombin time was found to be over one minute. A diagnosis of hemorrhagic disease of the newborn was made, and the child was given 60 mg. of vitamin K and 70 cc. of fresh blood intravenously. The prothrombin time then dropped to 15.6 seconds. He began to take some feedings, but on the sixth day severe and projectile vomiting recurred. This symptom, together with the evidence of bleeding from the upper gastrointestinal tract, suggested a diagnosis of acute peptic ulcer. X-ray showed the pyloric canal to be elongated, with definite antral spasm. He was given methantheline (Banthine) bromide, but did not improve, and, since obstruction was marked and he was losing ground, a surgical exploration was done with a view to doing a gastroenterostomy if an ulcer were found. There was malrotation of the intestine, the cecum lying high under the liver and bound down by dense adhesions. These were freed and the malrotation reduced. There was no gangrene of the bowel, no dilated or narrowed length of bowel, and no other anomaly found.

Following surgery the child continued to vomit severely, and on x-ray a dilated first and second portion of the duodenum were found with halting of the barium column in the third portion. The infant was operated on again, and a gastroenterostomy was performed. The bowel was again inspected, and no anomaly or circulatory disturbance was found. Again the child did poorly, continued to vomit greenish bile-stained material, and finally died in spite of vigorous supportive therapy.

Postmortem examination revealed a functioning gastroenterostomy, no evidence of peptic ulcer, and no mechanical obstruction. It is possible that there may have been a superficial ulcer which healed, but there were no findings to account for his death.

Congenital Absence of the Sternum. Dr. M. Andelman and Dr. S. Mackler.

This child was admitted to the hospital at the age of about 12 hours. The mother, 32 years of age, had had good prepartal care, and delivery was normal. When examined, the child appeared normal except for the obvious defect, i. e., complete absence of the sternum and a defect in the skin about the size of a half dollar, with protrusion of the pericardial sac.

Attempts to approximate the two sides of the thoracic cage produced cardiac embarrassment. It was therefore decided to use some type of graft, such as sternum from a baby who had died. This procedure was performed about 10 days after birth, and the postoperative course was uneventful except that the child failed to gain weight in spite of efforts to increase the caloric intake. This persisted until about one month postoperatively, and since then he has gained normally. The mother said that her first baby also gained poorly for the first six weeks of life.

The only case found in the literature in which the patient survived surgery for this condition was reported in 1949, and that child died a few months after surgery.

Posterior Mediastinal Mass in an Infant. Dr. Lawrence Breslow.

This was the third child of a white woman, born June 28, 1952, four weeks prematurely. The baby left the hospital at 2 weeks of age, having regained the birth weight, and did well at home for four or five weeks, at which time the weight was only 14 oz. (400 gm.) above that at discharge. At this time fluoroscopic examination revealed a posterior mediastinal mass extending into the right midlung field, confirmed by roentgenogram. The child was hospitalized and the roentgenographic examination was repeated with a barium swallow, which revealed displacement of the mediastinal structures to the left.

Four weeks later the mass was no longer seen on fluoroscopy or roentgenogram. There was no history of any episode of vomiting or choking during this interval. The child continued to do well, and at the age of 8 months the chest appeared to be completely normal on physical and laboratory examinations.

It can only be assumed that the child had either a bronchiogenic cyst which slowly evacuated itself or a reduplication of the esophagus with a fistula to the esophagus.

Dermatomyositis. Dr. Ralph H. Kunstadter and Dr. Allen Schultz.

A 10-year-old white girl entered Sarah Morris Hospital for Children with the history that in October, 1951, puffiness of the lower eyelids developed and she complained of constant fatigue. Redness of the skin around the fingernails, with some dryness and flaking, was also noted. In

January, 1952, redness of the skin of the face with a butterfly distribution and a reddish thick scaly rash over the elbows and knees developed. She was hospitalized in another city and treated with corticotropin (ACTH) followed by cortisone, with little improvement.

On admission, her temperature was 100.4 F.; pulse rate, 104; respirations 20; blood pressure, 108/68; height, 55 in. (140 cm.); weight, 67 lb. (30.4 kg.). She was well developed and pale and appeared chronically ill. Over the lower eyelids and upper face there was a heliotrope butterfly-shaped discoloration; slight eyelid edema was present. Over the lower abdomen a cluster of papulovesicles characteristic of herpes zoster was present. An erythematous, lichenified, and scaling eruption of both elbows and knees was noted, and slight redness, puckering, and scaling of the skin at the base of the fingernails was present. Physical examination otherwise showed nothing significant.

The blood examination showed the following: hemoglobin, 8 gm. per 100 cc.; red blood cell count, 4,200,000; white blood cell count, 6,350; differential count, segmented cells 78%, nonsegmented cells 3%, eosinophiles 1%, basophiles 2%, monocytes 3%, lymphocytes 14%. The erythrocyte sedimentation rate was 10 mm. in one hour and the antistreptolysin titer 50. The findings on electrocardiogram and chest plate were normal. Findings on urinalysis were normal, and blood chemistry tests were within normal limits. Bone marrow examination showed a nonspecific marrow pattern with increase in histiocytes and eosinophiles. The preparation for L. E. cells was negative. Muscle biopsy revealed typical histologic changes of dermatomyositis, lymphocytic infiltration, fragmentation of muscle fibers, edema, and waxy degeneration.

The child was treated with cortisone ointment on the rash for 10 days with no improvement. A month later edema of both legs developed, and the skin became shiny. The sedimentation rate was now 29 mm. in one hour. From Sept. 2 to Oct. 19 she received corticotropin with no improvement. Cortisone therapy was instituted until Nov. 21. Two weeks after completing this course she had some difficulty in climbing stairs and complained of increased stiffness of the extremities. There was no change in the rash. Spindle-like swellings of the fingers appeared.

Because of apparent failure of corticotropin and cortisone to alter the condition, *p*-aminobenzoic acid therapy was started, 1 gm. four times a day. When the child was last seen, on Jan. 2, 1953, the mother thought there was less swelling of the hands and feet and less redness of the face. A rough systolic murmur heard best at the third left interspace and some increase in cardiac size were present. The erythrocyte sedimentation rate was 38 mm. in one hour. A low-grade fever persisted. Administration of *p*-aminobenzoic acid is being continued for another month.

In spite of some clearing of the skin manifestations, the disease is apparently progressing as manifested by persistent low-grade fever, elevated erythrocyte sedimentation rate, increased muscle weakness and atrophy, and cardiac involvement.

Pituitary Disease. Dr. I. P. Bronstein.

The purpose of this presentation is to emphasize the difficulty in making a positive diagnosis of anterior pituitary disease; the anterior hypothalamus is included. The diagnosis of anterior pituitary disease is usually presumptive, and is also made by excluding other endocrine glands which may produce identical symptoms. Laboratory tests and adjuncts, including sugar and insulin tolerance and also hormonal assays, have proved equivocal. The anterior pituitary (anterior hypothalamus) may be responsible for three important symptoms which are uncommon, and their opposites which are rather uncommon. Frequently, we encounter undersize, adiposity, and undersexualism. So far as remarkable thinness is concerned, as seen in so-called Simmond's disease, we have never studied a true case. In 1949, Sheehan took issue with the whole concept and, as occurred with obesity and Fröhlich's syndrome, a change in our thinking is taking place. We were able to make an unquestionable diagnosis in a limited number of instances, either roentgenographically, by operation, or at necropsy.

Nasal Bleeding of Unusual Cause. Dr. A. Matheson.

The clinical course and autopsy findings of a lymphosarcoma of the nasopharynx in a 3½-year-old Chinese boy were presented. The outstanding symptom for several months before, and for two weeks after the tumor was recognized, was nasal hemorrhage. Anemia and cervical

lymphadenopathy were present late in the course of the disease. Evidence of metastases was not present until a week before death. Irradiation therapy dissolved the local tumor and local adenopathy, but did not prevent metastases.

The autopsy findings revealed extensive generalized metastases, including the heart, mediastinum, gastrointestinal tract, lymph nodes, pancreas, and kidneys. The kidneys were almost completely destroyed by nodular tumor masses. The characteristic invading cell on microscopy was a round cell with an oval nucleus, almost completely replacing the cytoplasm.

Glycogen-Storage Disease. Dr. J. Calvin.

In this patient an enlarged smooth liver was noted during the newborn period, and during the first week of life there was considerable vomiting and a 9-oz. (255 gm.) loss of weight. During the second and third week the infant remained well at home and gained 1 lb. (455 gm.). When 3½ weeks old, he vomited, became too listless to take his feedings, and was hospitalized.

When examined, he was very lethargic and did not move or cry. The liver was smooth and three fingers below the costal margin. Generalized convulsions began within 12 hours after refusing food. Several blood studies showed the blood sugar level to be around 10 mg. per 100 cc. Cholesterol was 350 mg. per 100 cc. Other values were within normal limits. The spinal fluid examination showed nothing significant. The urine contained 4 + acetone, but no sugar or albumin.

The symptoms of lethargy, coma, and convulsions could be explained on the basis of hypoglycemia. These were controlled, and the infant was revived by intravenous and subcutaneous administration of dextrose and Amigen (protein hydrolysate [Mead Johnson & Company]) solutions. An epinephrine tolerance test was performed after the liver, blood, and other tissues had been amply replenished, and the blood sugar was 80 mg. in 15 minutes. After the epinephrine was given the blood sugar was 60 mg. in 30 minutes; later 53 mg. and 46 mg. in 60 minutes. The infant was maintained on frequent feedings with a high-protein low-carbohydrate content (protein milk), supplemented at times with subcutaneous administration of Amigen and dextrose. There was moderate weight gain and the child seemed to be in fairly good condition.

However, when the child was 6 weeks old, two and one-half weeks after admission to the hospital diarrhea and vomiting occurred, he again became apathetic and refused feedings, and convulsions developed. The blood sugar at this time was 12 mg. per 100 cc. Von Gierke's disease or an islet cell tumor of the pancreas were considered as possible diagnoses. Exploratory abdominal surgery was decided upon, to be followed by corticotropin (ACTH) therapy. Unfortunately during operation there was respiratory and cardiac arrest lasting 12 minutes during anesthesia, followed by hyperpyrexia, coma, and death within 24 hours.

The postmortem examination revealed glycogen-storage disease involving principally the liver and kidneys, with moderate glycogen infiltration of the myocardium. Grossly, the liver was enlarged and soft in consistency, with rounded margins. The kidneys showed glycogen deposits mostly in the tubules and some in the capsular space.

George F. Munns, M.D., *President*

Joint Meeting with the Chicago Medical Society, Feb. 17, 1953

Clinical Evaluation of the School Health Program in the Chicago Public Schools: Panel Discussion

Cardiac Aspects of School Health. Dr. Carl J. Marienfeld.

Prior to 1951, the cardiac problem consisted primarily of special school placement where heart patients were, and for the most part still are, thrown together with orthopedically handicapped and spastic children under the general category of exceptional children. In these schools training in general is the same as in regular schools except that teachers are specially trained in dealing with orthopedic and spastic children, rest hours can be arranged, and ramps and elevators are available. Physical therapists, untraviolet therapy, and swimming therapy are also available. Periodic examinations are made by a school physician.

In 1951, Dr. Kenneth S. Nolan and the Chicago Heart Association, through Dr. Hugh McCulloch, reviewed all cases in the special schools, of which group 357 children were so placed by virtue of heart disease. It was found that many of these had remained in a special school for many years after a mild attack of rheumatic fever which left little or no heart damage. Many were able to assume full and normal activity even though a heart murmur persisted.

During the past year, in addition to the 150 old cases that were reviewed since May, there have been 83 new cases, the children in 34 of which were admitted to special school, 30 to regular, and 3 to a convalescent home; 15 were given home teaching. A child in the acute or subacute stage should not be in school, but should either have a home teacher or be in a convalescent home in which teaching can be given; unless there has been severe heart damage, regular school placement is to be preferred. The children now in special schools are those with long-standing or severe heart disease or those with congenital heart disease with reduced cardiac reserve.

There have been no great difficulties with children returned to regular school, although if a child should acquire scarlet fever or a streptococcic infection, there is danger of exacerbation of the disease. There is no way of preventing such infections in either the special or the regular schools, and the problem must be met by teacher and principal education. It is hoped that eventually every school will be covered by a teacher nurse, but in the interim the Chicago Heart Association has made available funds for a full-time teacher nurse for the cardiac problem, who will give attention to each child returned to school following an attack of rheumatic fever, and curb unnecessary activities.

The greatest problem facing the adolescent child is placement in the competitive world of adults. Even though his diseases may be mild, he has been in the hospital at various times and has carried the constant fear of death. When he leaves school he suddenly realizes that he is expected to take his place with nonhandicapped individuals in direct competition for jobs, for most of which he cannot pass the physical examination, plus the fact that in many instances he cannot buy insurance to help guarantee his future if he does get a job.

We have within the framework of the Board of Education vocational schools, counselors, and psychologists to help determine aptitudes and interests. The time to start rehabilitation is while the child is in school, through the educational channels already set up, and then in cooperation with adult rehabilitation units to continue until the child has obtained maximal benefit. At present, as a partial solution, a program has been worked out whereby the child is referred to the placement service wherein all these services are available and followed throughout his education.

The Dental Aspects of School Health. DR. JOHN C. BERGMANN.

The dental health education program was planned by a committee consisting of representatives of the Health Department, the Board of Education, and the various university dental schools of the city. Approximately 82,000 children in 153 public schools have had their teeth inspected since the program was started in September, 1949.

Members of the Chicago Dental Society participate voluntarily in the program. Also are included staff dentists of the Chicago Health Department, dental hygienists from Northwestern University and principals and classroom teachers in the various schools. Inspections are made by dentists and data so obtained are recorded as part of the health record of each child. Hygienists, in talking to children in each room of every school participating in the program, stress the four following fundamental health rules:

1. Brush the teeth immediately after each meal and before going to bed; if you cannot brush the teeth immediately after eating, rinse the mouth with water.

2. Eat an adequate amount of protective foods, such as milk, fruits, vegetables, eggs, meat, whole grain cereals, and bread. Between meals, children should be given fruit juices, fresh fruit, unsweetened dried fruit, nuts, popcorn, milk, or cheese, etc., rather than candy, gum, pop, or other sweets.

3. Children should have their teeth treated with sodium fluoride by a dentist.

4. Each child should visit the dentist as frequently as the dentist advises, usually at least twice a year.

Educational pamphlets are left with the classroom teacher, with instructions on distribution to parents.

The Chicago Health Department operates 48 dental clinics for indigent children, to which children are referred by various agencies, such as the Metropolitan Welfare Council, the Juvenile Court of Cook County, Children's Hospitals, school principals, and nurses.

During the school year 1951-1952, the following services were rendered:

Treatments, consisting of prophylaxis, anodyne, treatment of abscesses, sodium fluoride, zinc oxide, and eugenol	30,633
Fillings	55,302
Extractions of primary and permanent teeth	12,697
Mouth surveys	24,984
Educational lectures	2,409
Total operations or services	126,025

Hearing Aspects of School Health. DR. RICHARD E. MARCUS.

A hearing conservation program places emphasis on finding children with hearing loss and seeing that proper therapeutic and rehabilitative management is realized. The otolaryngologist is interested in cooperating with others in the community to establish a hearing conservation program and to participate in it.

The principles of the Chicago Hearing Conservation Program are (1) organization of a planning committee with representatives from the Chicago Medical Society, the Chicago Laryngological and Otological Society, the Board of Education, the Chicago Hearing Society, and citizens' school groups; (2) technical organization for individual and group pure-tone audiometry, using trained audiometrists who follow a predetermined pattern of testing; good screening should identify those who have normal hearing, whereas those failing to meet the minimum requirements of the screening are investigated further by individual pure-tone audiometry; (3) establishment of otologic screening clinics for children found to have a hearing loss of 30 db. or more, in more than one frequency in either ear; tentative diagnoses are made in those clinics, and the need for further care, where indicated, is explained to the parents; (4) adequate follow-up to ensure the therapeutic and rehabilitative management, in private or public hands; (5) educational placement of the children on an individual basis, depending on the type of hearing loss, laterality, prognosis, and duration. The previous or possible future effects of the hearing loss on the child's speech and school progress are also considered.

From September, 1950 to February, 1952, 49,574 children were screened audiometrically (an average of 4,000 per month), of whom 2.5% were found to have a hearing loss of 30 db. or more, in more than one frequency in either ear. Of the children found to have a hearing loss, 85% appeared at the otologic screening clinics. Of those who appeared, 84% had a hearing loss, in 63% of whom it had not been known. On a six-month recheck of 304 children for whom medical care was recommended and carried out, 58% showed improvement, 34% showed no improvement, and 8% were worse. Where medical care was not carried out, 32% improved, 56% showed no improvement, and 10% were worse. Of children referred by teachers from grades other than those being screened and from ungraded classes 5.5% had actual loss. It is noteworthy that where medical care was carried out, 6 out of 10 children showed improvement. Where medical care was recommended but not carried out, only 3 out of 10 showed improvement. Also, where medical care was not recommended, 6 out of 10 children showed improvement and, in general, the percentages in this group are almost exactly the percentages in the group in which medical care was carried out. With proper disposition, therefore, it seems that 6 out of 10 children will show improvement in hearing.

In spite of the fact that the children referred by teachers from grades other than those being screened and from ungraded classes had been sent specifically because they were suspected of having hearing loss, only 5.5% actually were found to have such a loss. This illustrates that hearing loss is difficult to observe grossly.

School Health Councils. DEPHANE A. JENSEN, R.N.

In January, 1952, the first nine teacher-nurses were assigned to 17 of the Chicago Public Schools, and since that time a public relations program has been set up to acquaint the faculty and communities with the service. This service begins with the health appraisal of the individual child. The importance of the physician-patient relationship is emphasized, or, in the case of

the medically indigent, an individual plan is worked out following a review of the child's health history and immunization status. The cumulative health record follows him through his school life.

Routine screening programs as to height, weight, and nutrition problems have been set up, and education on these has been carried out in various ways, with satisfactory participation by both teachers and pupils. Following this, hearing and vision are screened, under the direction of a technician from the Hearing and Vision Conservation Program, who trains as many teachers as is deemed necessary. The nurse's part is to coordinate the service and to explain the findings of the tests to the parents and recommend ophthalmological or otologic care as necessary. All children are referred to the family dentist annually. The record on the dental program has been encouraging, largely because of the efforts of the classroom teachers. Indigent children are referred to the Health Department clinic.

The Health Council attempts to coordinate the services of all groups interested in the school, and consists of representatives of school administration, teachers, medical and dental consultants, Health Department, Parent-Teacher Association, engineers, special teachers, teacher-nurses, truant officers, and any other specialists that might help with a current problem. The following purposes have been set up: (1) to stimulate cooperative responsibility in health and safety needs; (2) to promote interest in health among parents, teachers, and children; (3) to assist in providing healthful school living conditions; (4) to promote the coordination of school health services with education; (5) to stimulate interest in establishing conditions conducive to good mental health, and (6) to acquaint the home and community with the total school health program. The principles of the Health Council have been approved by the School Health Committee of the Chicago Medical Society.

Provision is made through a central file for contacting parents or physicians in emergencies. First aid supplies are available to all teachers. In the communicable disease program basic immunizations and boosters as recommended are encouraged. Emphasis is placed on early exclusion and medical care rather than screening on readmission to school.

The Parent-Teacher organization has aided in interpretation of the health program to parents and community groups and in emphasizing the fact that the primary responsibility for the health of children rests with the parents.

At the present time there are 14 teacher-nurses covering 30 schools and, it is hoped, there will be expansion to cover the entire school population and thus the optimum physical and mental health be obtained for every child in the Chicago Public Schools.

The School Health Program: Evaluation. DR. KENNETH S. NOLAN.

A complete school health program encompasses many phases beyond the scope of the present discussion, listed under two general categories—health promotion and health appraisal. Health promotion includes general measures relating directly to the child's health or instruction during school hours; included are safety control, environmental sanitation, school lunch program, recreation, health instruction, physical education, and psychologic testing. The Chicago Public Schools have long had separate divisions specifically devoted to each of these areas, prior to the establishment of a general program.

Basic premises agreed upon by the Bureau of Health Services of the Board of Education in the initial planning for any procedures for health appraisal of children were as follows:

1. Parents have primary responsibility for the health of their children, and the school services should be designed to assist parents in discharging this responsibilty, but not to assume it for them.

2. All health appraisal measures which stem from school sources should be directed at the detection of those defects which present the greatest likelihood of impeding a child's school progress or adjustment. Moreover, such detection is most beneficial if it occurs early in his school career. Hence, the younger age groups receive first priority.

3. Special attention should be devoted to assuring the follow-up for needed care if the initial phase of health appraisal is to be at all worth while.

Emphasis has been placed on a physical examination for the child who enters school for the first time. The results have been highly gratifying to date as follows:

	Sept., 1951	Feb., 1952
New registrants enrolled	26,138	16,675
Completed medical records returned	11,782 (45%)	9,434 (57%)
Examined by family physician	11,160	8,823
Examined in clinics without cost	488	535

These figures compare favorably with other major school systems.

Recent figures from the program of vision screening are summarized as follows: children tested, 133,126; below minimum requirements, 21,466 (16.1%). An analysis of the forms returned by ophthalmologists who examined these children indicates that 90% of the school referrals were valid. However, only about 38% of all children referred are definitely known to have had complete eye examinations as recommended. An incidental fact is that 96 children discovered by the visual screening program had visual acuity so impaired that transfer to a sight-saving class was recommended.

Additional screening programs include periodic chest x-rays of high school students and Board of Education employees, through the facilities of the Tuberculosis Institute of Chicago and Cook County. From November, 1951, through May, 1952, 25,066 students had chest x-rays, and 108 (0.3%) suspect cases of pulmonary tuberculosis were reported.

The heart of the entire program lies with the activity of the trained public health nurse employed to serve the schools as a teacher-nurse. An effective health service program, reaching into all schools and making possible the efficient utilization and coordination of all existing community health resources for children, must await the augmentation of the limited numbers of such trained personnel.

Obituaries

PHILIP VAN INGEN, M.D.
1875-1953

In the passing of Philip Van Ingen, on March 28, 1953, pediatrics has lost one of its cornerstones.

As a boy he attended the Cutler School in New York and the Hill School at Pottstown. A graduate of Yale (A.B.) in 1897, he obtained his M.D. degree from the Columbia University College of Physicians and Surgeons in 1901. He served

PHILIP VAN INGEN, M.D.
1875-1953

an internship at the Presbyterian Hospital in New York for two years and a residency in pediatrics at the New York Foundling Hospital. Postgraduate studies were done in Vienna before he started to practice in New York in 1905. He was soon appointed to the staff of the Willard Parker and the Bellevue Hospitals, and in 1913 was appointed an assistant pediatrist at Roosevelt Hospital, becoming an

attending pediatrist in 1925. He held the position until 1938, when he relinquished his duties at the hospital and became a consultant. He was a clinical professor of pediatrics at Columbia from 1920-1925.

He amassed about all the honors which could fall to one man. He was a founder member of the American Academy of Pediatrics and became its president in 1937. He was also a president of the American Pediatric Society and of the American Child Health Association. He was one of the nine members of the original Board of Pediatrics in 1932 and served for six years.

He was chairman of the Committee on the Medical Care of Children of the White House Conference on Child Health and Protection, which ex-President Herbert Hoover organized in 1929. He collaborated with the late Dr. Hamill and the late Dr. Blackfan in the preparation of the 1930 White House Conference.

When World War I was declared, he was one of the first to enlist, with the rank of captain. Later he was promoted to lieutenant colonel in command of Field Hospital #331, serving in France and Italy. For meritorious services he received la Croce al Merito di Guerra.

One of his greatest services and contributions was to the New York Academy of Medicine. The last active years of his life were spent in the compilation and the publishing of a history entitled "The New York Academy of Medicine, Its First Hundred Years." He devoted, with high enthusiasm, two years of his life to the preparation and documentation of this book. One of its most interesting chapters is on Abraham Jacobi, when he was president for two terms, from 1885 to 1888. On the part of Philip Van Ingen this book represents a labor of love. He has left the New York Academy of Medicine an imperishable record of its contributions to public health and welfare and to medical education, national and international in scope. He has brought to life, in this interesting chronicle, the needs and services which many distinguished physicians have rendered over the past 100 years.

But, when all is said and done, his real love was for children, and to them he dedicated his life. In his latter days his great interest was for Roosevelt Hospital, where he had given so many years of his life. Here he had made some of his closest friends, and he never lost his identification with them or the institution. In his services to the hospital and to the children under his care, he gave meaning to the motto inscribed on the stone in front of its door, "Qui plantavit curabit." These words will remain Philip Van Ingen's greatest glory, and for these we will best remember him.

Abstracts from Current Literature

Metabolism; Infant Feeding; Milk and Other Foods

Factors of Importance in Breast Milk. Theresa B. Haddy and Forrest H. Adams, J. Pediat. **40**:243 (Feb.) 1952.

Haddy and Adams review the well-known advantages of breast milk in infant feeding. Nicotine is excreted in breast milk, but, in the case of the mother who smokes a moderate number of cigarettes a day lactation is not diminished and the infant has no adverse effects. Phenobarbital should be given in sedative ½ grain (30 mg.) doses rather than in hypnotic 1½ grain (0.10 gm.) doses, since it is excreted in breast milk and may affect the nursing infant. Bromides should be avoided by the nursing mother, as should heavy metals, such as lead, mercury, and arsenic.

Macdonald, Pittsburgh.

Serum Proteins of Infants Under Normal and Pathological Conditions; Analysis by Electrophoresis. C. Imperato, Lattante **23**:321 (June) 1952.

A chart is presented showing the range in the amounts of the serum proteins—albumin, alpha globulin, beta globulin, and gamma globulin—in 63 blood specimens obtained (1) from the umbilical cords, (2) from infants 4 to 42 days old, (3) from infants 1½ to 5 months old, and (4) from infants 6 to 25 months old. Use of this chart as representing normal figures affords a comparison with the results of the differential protein analyses of the sera of 10 infants with various infections.

Higgins, Boston.

Therapeutic Value of Pantothenic Acid in Childhood. Wolfgang Ehrengut, Arch. Kinderh. **143**:140 (No. 3) 1951.

The author reports favorable results with pantothenic acid (75 mg. three times a day) in cases of hypothyroidism. Administration is required over a period of several months, preferably in combination with thyroid tablets. The treatment has a good effect upon metabolism, loss of hair, and mental condition.

Gottstein, Chicago.

Protein Deficiency in Infantile Dystrophy. R. Thurau, Monatsschr. Kinderh. **99**:241 (July) 1951.

So-called infantile dystrophy, also called marasmus of infancy, often attributed to malnutrition per se by some authors, was found to be due to chronic infection involving especially the small intestine and thus leading to hypoproteinemia.

Weight, chest circumference, and body surface were below normal in the infants with this condition. Hemoglobin values were only slightly decreased during the first month of life but fell during the third and fourth months. Hemodilution was found during the first month, while plasma volume was increased. Plasma proteins fell during the second month of life, especially the albumin fraction. During the next few months there were decreased values for both albumin and globulin, including the gamma globulin fraction.

Hartenstein, M. C., U. S. A.

Hygiene; Growth and Nutrition; Public Health

Growth of Thiocyanate Space in Infancy and Childhood. Robert S. Ely and Wataru W. Sutow, Pediatrics **10**:115 (Aug.) 1952.

The thiocyanate space of 108 normal subjects was determined and the blood volume of 50 of these. These values are presented with relationship to physical indices of growth.

The decrease in thiocyanate space as per cent of weight is shown to occur primarily in the first year of life. The significance of this change is discussed with relation to total water and per cent of body fat.

From the Authors' Summary.

Prematurity and Congenital Deformities

Retrolental Fibroplasia: Blindness in Infants of Low Weight at Birth. Alfred Appelbaum, California Med. **77**:259 (Oct.) 1952.

Retrolental fibroplasia is today the principal cause of blindness in children of preschool age, exceeding all other causes combined. The disease occurs in infants of low weight at birth, commonly those born prematurely. The incidence of the disease is rising at an alarming rate. Vitamin E deficiency, corticotropin (ACTH) deficiency, use of cow's milk in place of mother's milk, and improper oxygenation have been suggested as etiologic factors, but the cause remains a mystery. Often the incidence is high in institutions in which maximal care is given premature infants.

Clinically, the disease advances through an "active," phase during which regression is possible and a "subsiding" or "cicatricial" phase, which terminates with the formation of a disorganized opaque mass behind the lens. The earliest manifestations are noted in the fundi. Hemorrhages, neovascularization, transudation commencing in the periphery, and retinal separation contribute to the formation of the characteristic retrolental membrane. The diagnosis may be made when the retrolental membrane is observed in the eye of an infant whose weight at birth was low. Differential diagnosis is required occasionally.

Thus far no form of therapy has prevented or reversed the pathologic changes successfully. Use of vitamin E, corticotropin, and mother's milk has not influenced the incidence of the disease. Avoidance of premature delivery, if possible, is indicated.

Pryor, Redwood City, Calif.

Bromsulphalein (BSP) Excretion Test as Measure of Liver Function in Premature Infants. William Obrinsky, Malcolm L. Denley, and Ralph W. Brauer, Pediatrics **9**:421 (April) 1952.

In normal adults, following a single intravenous injection of sulfobromophthalein, the logarithmic decline of plasma sulfobromophthalein levels is rectilinear. In patients with hepatic disease, the plotted values are curvilinear, with flattening of the curve, or "saturation," occurring at high plasma sulfobromophthalein concentration. The use of the excretion curve provides a dynamic representation of the hepatic uptake of dye throughout the test, avoids the theoretic objections to the 45-minute test, and obviates the necessity for plasma volume determinations.

Thirty-five healthy premature infants of varying age and weight were the subjects of a study of liver function by the sulfobromophthalein sodium excretion test. As measured by this test, there is progressive maturation of liver function with age in premature infants that does not necessarily correlate with weight. The excretion curves obtained on premature infants 18 to 28 hours of age resemble those of older patients with severely impaired hepatic function. Clearance of dye in these subjects is slow throughout the test. With increasing age there is a gradual change in the pattern of the curves, characterized by the appearance of the initial sharp slopes coincident with rapid dye removal. "Saturation," or flattening of the curves, persists until a later age and takes place at progressively lower plasma levels. This biphasic pattern is apparent in premature infants over 4 days of age. The normal adult pattern is not attained until about six to eight weeks although the 45-minute-retention values become normal at three weeks.

The gradual development of normal excretion curves in premature infants may be explained by the progressive maturation of the anatomic and physiologic factors involved in the mechanism of sulfobromophthalein clearance. The increasing size of the capillary network in the liver that occurs with advancing age in effect increases hepatic blood flow and exposes a greater volume of blood in the sinusoids to the activity of the liver. The appearance of the initial sharp slopes, representing rapid dye removal, probably results from this increasing vascularity and possibly from maturation of the hepatic uptake mechanism. The continuance of the biphasic character of the curves probably results from inability to excrete the dye efficiently into bile.

From the Authors' Summary

Effects of Early Feeding of Strained Meat to Prematurely Born Infants. M. B. Andelman, P. S. Gerald, A. C. Rambar, B. M. Kagan, Pediatrics **9**:485 (April) 1952.

This study was undertaken to determine the effect on prematurely born infants of the feeding of strained meat. One group of infants received a standard formula; another, an identical formula to which strained meat was added, and a third group was breast-fed.

Meat added to the diet of prematurely born infants weighing between 2,000 and 6,750 gm. was well accepted. There was no significant difference in the symptoms or signs of illness among the infants of any of the three groups.

It was found that a plot of the body weight of such infants against age generally gives a smooth sigmoid curve. The central portion of the "S" forms a remarkably straight line. The linear portion of the curve is located in the interval between 2,500 and 4,500 gm. body weight. The location is relatively unaffected by either age or birth weight. The slope of this linear portion was taken as the measure of the weight gain. A similar relationship was found for the crown-heel length. These findings are not only of academic interest but also of practical importance, since they provide an unusually good tool for further nutritional and other studies.

Growth as measured by rate of change of weight and length did not differ significantly between the experimental group and the control group. The breast-fed group exceeded the experimental and control groups in both weight and length gain. The greater weight gain in the breast-fed group as compared with the experimental group was statistically significant.

Minimum values for red blood cell count and hemoglobin were observed at 12 weeks of age. These values rose steadily until about 20 weeks of age, at which time they became relatively stable. There was no significant difference in the hemoglobin concentration or red blood cell count among the three groups at 12 weeks of age. At 20 weeks, however, the meat-fed, or experimental group, exceeded the control group by a significant amount. The mean difference in hemoglobin concentration of the experimental group and that of the control group was 1.0 gm. per 100 cc. This finding is of especial interest, since the experimental and control groups both received more than adequate amounts of iron added to the diet.

A gradual rise in serum protein concentration was noted with increasing age. At 12 weeks of age, the serum protein concentration for the breast-fed group was significantly lower than that for the experimental group, the average difference being 0.40 gm. per 100 cc. The average difference between these two groups at 20 weeks was 0.35 gm. per 100 cc., but there were too few in the breast-fed group at this time to give the difference statistical significance. The serum protein concentrations in the experimental and the control groups did not differ significantly at either 12 or 20 weeks of age.

Analysis of the total volume intake of the formulae showed that intake of the experimental group was reduced in proportion to the greater caloric content of the meat-containing formula. The result was that the daily intake of calories was the same in both the experimental group and the control group. This remarkable reduction in volume of intake by prematurely born infants in the weight groups concerned and the strikingly uniform caloric intake are of interest particularly in view of current thoughts on self-selection of diet.

No differences were noted in the weight or length gain, hemoglobin, red blood cell count, or serum protein concentration between the experimental group and the control group when the infants reached one year of age. From the Authors' Summary and Conclusions.

Newborn

Bronchoscopic Problems in the Newborn. Paul H. Holinger and Kenneth C. Johnson, Illinois M. J. **101**:68 (Feb.) 1952.

According to the authors, bronchoscopy of newborn infants may be indicated for aspiration of secretions but when a bronchoscopic examination is necessary, usually other severer pathology is present. This consists generally of a congenital anomaly.

An examination of the infant may reveal certain signs of great value in localizing the point of obstruction. Besides a careful history and physical examination of the chest, the character of

the fontanel and the deep reflexes are of significance in determining the possibility of cerebral birth injury. One of the most important examinations is an x-ray study of the chest and neck.

One should always be prepared to do a tracheotomy before doing bronchoscopy on a newborn infant, not because of complications due to the bronchoscopy but because of the pathology which necessitated the bronchoscopy. Contraindications for bronchoscopy on the newborn infant are cerebrovascular accidents and pneumothorax.

BARBOUR, Peoria, Ill.

EFFECT OF APNEA NEONATORUM ON INTELLECTUAL DEVELOPMENT. GENE L. USDIN and MARVIN D. WEIL, Pediatrics **9**:387 (April) 1952.

The intelligence of a group of 41 children who had been apneic for three or more minutes at birth was compared with that of a control group of 43 children who had breathed spontaneously at birth. The children were 13 and 14 years old at the time of the study. All the children had been born in the Cincinnati General Hospital in 1937 and therefore should derive from an essentially similar socioeconomic strata. So far as seemed possible, all cases with factors that might in themselves have impaired the child's motor or intellectual development were excluded. Individual intelligence tests were administered. A statistical analysis of the results of these tests revealed no significant difference between the intelligence of the apneic and that of control group.

FROM THE AUTHORS' SUMMARY.

CONGENITAL FAMILIAL NONHEMOLYTIC JAUNDICE WITH KERNICTERUS. JOHN F. CRIGLER JR., and VICTOR A. NAJJAR, Pediatrics **10**:169 (Aug.) 1952.

Seven patients with congenital familial nonhemolytic jaundice with kernicterus are presented. Six of these were hospitalized and studied. For one the diagnosis was made from the history. The abnormality appears to be genetically determined, being inherited most likely as a lethal recessive characteristic. The only abnormal laboratory findings are pronounced elevation of indirect serum bilirubin and delayed excretion of injected bilirubin. Pathologically, bile thrombi in the canaliculi were found in all patients studied, with very slight periportal fibrosis present in the liver sections from cases 1, 5, and 6. In the single brain examined (Case 2), kernicterus was described. The prognosis is extremely poor, the patients generally dying in the first year of life. No therapeutic measures have been found helpful. The similarity to a previously described disease, familial nonhemolytic jaundice, is discussed.

FROM THE AUTHORS' SUMMARY.

INTESTINAL OBSTRUCTION IN THE NEWBORN INFANT DUE TO AGENESIS OF THE MYENTERIC PLEXUS (CONGENITAL MEGACOLON). WILLIS J. POTTS, JOSEPH D. BOGGS, and HARVEY WHITE, Pediatrics **10**:253 (Sept.) 1952.

Agenesis of the myenteric plexus or congenital megacolon often manifests itself by intestinal obstruction in the newborn infant. Medical management in such cases is not effective. If the infant's condition warrants, immediate resection of the neurogenically abnormal bowel should be done. In the poor-risk infant sigmoidcolostomy is the procedure of choice.

FROM THE AUTHORS' CONCLUSIONS.

ICTERUS NEONATORUM. DUONG BA BANH and NGUYEN NGOC SAN, Presse méd. **59**:892 (June 23) 1951.

This report is based on 50 cases of icterus of the newborn. The authors confirmed the findings of previous investigators, and concluded that icterus of the newborn is caused primarily by red blood cell destruction and hemolysis. This hemolysis can be increased by a number of factors, e. g., lowered resistance from prematurity or obstetrical trauma, hematoma formation, chilling, increased cell fragility, or perhaps an impairment of liver function, since bilirubinemia is increased. The authors found no relationship between incompatibility of blood groups in the family and appearance of icterus.

DAFFINEE, Melrose, Mass.

DEVELOPMENT OF THE INTESTINAL MUSCULATURE OF THE FETUS AND ITS CHRONOLOGICAL AND FUNCTIONAL RELATION TO THE PRESENCE OF MECONIUM. G. C. POLLIFRONI, Lattante **23**:331, 1952.

Descriptions and illustrations of the development of the intestine are based on sections from human embryos 5, 15, 19, 28, 29, 40, 53, 90, 110, 150, 190, and 210 mm. long. Meconium was first observed in the intestinal lumen about the fourth month (110 mm. embryo); intestinal musculature did not develop to the functional stage until one to two months later. The intestinal glands began to function at about the third month.

HIGGINS, Boston.

MORBID ANATOMY OF ERYTHROBLASTOSIS FETALIS AND ITS RELATION TO THE RH-FACTOR. S. RANSTRÖM, Acta paediat. **40**:41 (Jan.) 1951.

An autopsy series of 13 cases of erythroblastosis fetalis is reported. In this series there was an increase of the adrenal weight, premature differentiation of eosinophile and basophile cells of the hypophysis, insular hyperplasia (four cases), and increase of interstitial insular cells without islet formation (four cases) in the pancreas. The morphological changes are discussed. The two assumptions that the endocrine disorders are a manifestation of the adaptation syndrome and that they are the reason why the clinical symptoms do not begin till birth or afterward would reconcile the Rh theory of the etiology of erythroblastosis fetalis with the pathological anatomy and the clinical course of the disease.

FROM THE AUTHOR'S SUMMARY.

Acute Contagious Diseases

INCIDENCE OF POLIOMYELITIS: THE EFFECT OF TONSILLECTOMY AND OTHER OPERATIONS ON THE NOSE AND THROAT. ALDEN H. MILLER, California Med. **77**:19 (July) 1952.

A statistical survey was made of all the cases of poliomyelitis occurring in all of Los Angeles County during the three years of 1949, 1950, and 1951 in an attempt to determine the effect of operations on the nose and throat on the incidence of poliomyelitis. Tonsillectomy and adenoidectomy were the only operations noted with any degree of frequency. Yet, of the total of 3,601 cases of poliomyelitis that occurred in this three-year period there were only 20 (0.55%) in which the patient had had recent tonsillectomy and adenoidectomy.

The incidence of this disease in patients who had had tonsillectomy and adenoidectomy was compared with the "expected" incidence as determined from the incidence among other patients in the same age group. There was no significant difference between actual and expected incidence even during the summer months, when most cases of poliomyelitis occurred. The same was true with regard to recently tonsillectomized patients in the epidemic months of July through October.

In a separate survey of 675 patients with poliomyelitis, it was noted that only 30% ever had had tonsillectomy or adenoidectomy. Inasmuch as it is estimated that one of every three persons in the general young population nowadays has had tonsillectomy and adenoidectomy, this figure is no more or less than could be expected.

PRYOR, Redwood City, Calif.

POSSIBLE HOME CARE IN POLIOMYELITIS. MARTIN MILLS, California Med. **77**:29 (July) 1952.

Three main problems in providing adequate home care must be solved: (1) the obtaining of necessary equipment for the patient, such as bed, chair, hot-pack materials, appliances, and exercising apparatus; (2) the training of some responsible member of the family in the care that is required for the patient, and (3) the provision of adequate supervision by the physical therapist and the physician. The provision of these needs requires proper organization of all the persons and agencies in the community that have a contribution to make toward this care.

PRYOR, Redwood City, Calif.

PROGNOSIS OF PALSIES FROM CRANIAL NERVE INVOLVEMENT IN POLIOMYELITIS. HANS U. ZELLWEGER and E. GABATHULER, Pediatrics **10**:127 (Aug.) 1952.

Seven-hundred thirteen cases of poliomyelitis with spinal paralysis or encephalitic or bulbar symptoms are reviewed. The mortality rate in bulbar cases was four times that in spinal cases, whereas early recovery after bulbar symptoms was twice that after spinal symptoms.

A follow-up study of 59 patients with 69 palsies from cranial nerve injury performed 2 to 18 years after the acute stage showed that 39 patients with 48 palsies were cured; 18 patients had together 19 palsies of minor degree. Two patients, one with involvement of the sixth and one with involvement of the seventh cranial nerve, had severer sequelae. None of the palsies affected the lives of the patients.

Thus if patients with cranial nerve involvement stay alive, the late prognosis is much better; the majority of the palsies disappear, and the remaining ones have no crippling effect for the patient.

From the Authors' Summary.

Fulminating Meningococcemia Treated with Cortisone: Use of Blood Eosinophil Count as a Guide to Prognosis and Treatment. Horace L. Hodes, Ralph Moloshok, and Milton Markowitz, Pediatrics **10**:138 (Aug.) 1952.

The total blood eosinophile count has been studied as an indicator of the adequacy of the adrenocortical response to the stress of acute infection. Marked eosinopenia was found in seven children on the first or second day of acute meningococcal infection of mild or moderate severity and in 18 other patients with rubeola, pneumonia, or diphtheria. During convalescence the number of eosinophiles in the blood became normal. An eighth patient, who was admitted during the recovery stage of mild meningococcal infection, exhibited a normal count, which decreased sharply after the administration of corticotropin (ACTH).

In three patients admitted with fulminating meningococcal infection, initial eosinophile counts of 75 per cubic millimeter was found in one, and 130 per cubic millimeter in the other two. The eosinophile count response to the administration of corticotropin did not return to normal until the third week in the second case. These findings lend support to the belief that adrenal cortex function may be impaired in patients with overwhelming meningococcal infection.

Cortisone was employed in addition to usual forms of therapy in the treatment of three children with fulminating meningococcemia. One patient died shortly after admission, and the other two patients survived. It is believed that cortisone was of value in the latter two cases and that one of these patients would have certainly died without its use. It is suggested that the circulating eosinophile count may be of value as a guide in the prognosis and treatment of meningococcal infections.

From the Authors' Summary.

Differential Diagnosis of Aparalytic Poliomyelitis. G. Fanconi, Schweiz. med. Wchnschr. **81**:1070 (Nov. 3) 1951.

Cases of aparalytic poliomyelitis which run their course without any symptoms cannot offer any problem of differential diagnosis at the time. The author distinguishes a specific initial stage of the disease lasting 36 to 42 hours, followed by an afebrile period of one to five days. A distinction is made from the so-called first disease, which prepares the way for the invasion of the virus. A preparalytic stage, with meningeal, or perhaps better encephalomyelitic, symptoms, followed by adynamic symptoms, may lead to the paralytic stage. It is stressed that the presence of adynamic symptoms makes the diagnosis of poliomyelitis very probable. These symptoms are characterized by general muscular weakness, manifesting itself in various ways, by coarse tremor, and by muscular discomfort. Various forms of nonpurulent meningitis are tabulated, and the features differentiating these forms from poliomyelitis are discussed. Another tabulation lists in somewhat more detail various forms of virus meningitis. Special attention is called to the possible confusion of a harmless virus meningitis with a recurrence of streptomycin-treated tuberculous meningitis. Three occurrences of this kind are reported. For further details it is advisable to consult the paper.

Amberg, Rochester, Minn.

Pathogenesis of Encephalopathy Following Rubeola. Friedrich E. Günther, Arch. Kinderh. **144**:152 (No. 2) 1952.

One is not justified in assuming that almost all congenital malformations are of bacterial or viral origin, contracted during early pregnancy. Genetic factors mostly prevail in the etiology.

In the case of German measles, it is probable that the virus has a specific affinity for the cells of the ectodermal and mesodermal germ layers. The symptomatology reveals that the extra pyramidal, diencephalic, and vestibulocerebellar regions are involved sites of predilection.

The author discusses only the encephalic, not the cardiac, anomalies.

GOTTSTEIN, Chicago.

Acute Infectious Diseases

CAT SCRATCH SYNDROME. WILLIAM J. WALTERS, SEYMOUR S. KALTER, and JOHN T. PRIOR, Pediatrics **10**:311 (Sept.) 1952.

The clinical, laboratory, and pathologic findings of a series of cases of cat-scratch syndrome have been reviewed. In spite of a variable clinical course, certain features associated with a selected group of laboratory tests appear to be constant enough to be of diagnostic value.

A history of contact with a cat and/or scratch which is usually associated with a peripheral skin lesion, lack of lymphangitis, and presence of regional lymphadenopathy with tenderness to palpation are the most constant clinical findings. Fever, frequently emphasized as a characteristic clinical sign, may be extremely variable in type and duration or entirely absent. A skin test with cat-scratch antigen has been positive in all the authors' cases. If this antigen is not available, a negative Frei skin test in conjunction with a positive complement fixation test (Lygranum C. F.) is suggestive evidence for the diagnosis.

With positive evidence from the above data, biopsy of an affected lymph node with its relatively nonspecific pathologic picture is not considered essential for the establishment of the diagnosis of cat-scratch syndrome.

FROM THE AUTHORS' SUMMARY.

CONSERVATIVE TREATMENT OF PERITONITIS DUE TO PERFORATION ON THE COURSE OF TYPHOID FEVER. G. ROBERTO BURGIO, Pediatria **60**:274, 1952.

Four children and one adult with peritonitis due to a perforation of the intestine during the course of typhoid were treated medically. One child died, but the other four patients had a complete recovery. The medical treatment consisted in (1) combating shock by means of transfusion and intravenous injection of dextrose and saline solutions, (2) chemotherapy with streptomycin, sulfathiazole and penicillin, and (3) prolonged fasting. The percentage recovery by this treatment was much higher than has been reported following surgical intervention.

HIGGINS, Boston.

CONGENITAL MALARIA. E. A. VOSS, Arch. Kinderh. **143**:113, (No. 3) 1951.

The author reports two cases of congenital malaria. Intrauterine infection of the fetus has been proved by Eckstein. Though the normal placenta is impermeable, chills, fever, and hemorrhages of the affected pregnant mother favor infection of the embryo, which probably takes place during the delivery, since massive intrauterine infection would inevitably result in the death of the fetus. Morbid signs do not appear until one week after delivery and one treatment with quinacrine (Atabrine) would suffice.

GOTTSTEIN, Chicago.

TREATMENT OF OXYURIASIS WITH ATRIMON. NORBERT BROCK, ALBERT ERHARDT, and HILMAR WILMANNS, Deutsche med. Wchnschr. **77**:240 (Feb. 22) 1952.

The active principle of Atrimon is the carbinol base of methylrosaniline chloride. Its activity against the enterobiasis of the rabbit is equal to that of methylrosaniline chloride, while its toxicity tested in mice, rats, and rabbits is considerably less. The carbinol does not irritate the mucosa of the intestines. In strongly acid stomach contents the carbinol is restored to the dye. Therefore Atrimon is given with enteric coating. In the intestines, depending on the pH of the contents, not more than 10% is changed to the dye. Furthermore, the carbinol is not soluble in water. Atrimon has been used successfully in oxyuriasis of children and adults.

AMBERG, Rochester, Minn.

Chronic Infectious Diseases

Occurrence of Tubercle Bacilli Resistant to Streptomycin in Children with Tuberculosis. Virginia N. Wilking, Rosa Lee Nemir, Edith M. Lincoln, and Josephine D. Martin, Am. Rev. Tuberc. **66**:63 (July) 1952.

The occurrence of emergence of resistance to 10 or more micrograms of streptomycin was tested on 152 tuberculous patients, all of them under 13 years of age. In all, 387 cultures were tested; 46 of these (obtained from 17 patients) yielded organisms resistant to the above concentration. Seven resistant cultures were obtained from five patients not previously treated with streptomycin.

These 17 patients could be divided into two major groups: one in which an obvious correlation existed between the laboratory and clinical findings; the other in which a correlation could not be made. As Alexander and others have previously shown, the size of the inoculum was the most important single factor in determining the emergence of resistance. Only three patients showed the emergence of resistance; two of these died, and the other failed to improve with therapy. The other 14 did well clinically, despite the occasional appearance of resistant organisms in isolated cultures. These children showed an inconsistent laboratory response to streptomycin, but their clinical course was satisfactory.

Royce, Pasadena, Calif.

Primary Tuberculous Inoculation of an Ear Lobe: Report of an Unusual Case and Review of the Literature. Laura Grey Morgan, J. Pediat. **40**:482 (April) 1952.

A primary tuberculous lesion developed in an 18-month-old child at the site of an ear-ring puncture of an ear lobe. The primary lesion ulcerated. Regional lymphadenopathy developed. Treatment with streptomycin and paraaminosalicylic acid was successful.

Macdonald, Pittsburgh.

Tuberculosis of Cervical Lymph Nodes. Orvar Swenson and Wilfred T. Small, Pediatrics **10**:131 (Aug.) 1952.

Three of 11 patients with cervical tuberculous adenitis were treated with streptomycin preoperatively. For two of these patients, the results were complete failures, and surgery was subsequently required for control of the disease. One patient treated with streptomycin had a chronic draining sinus, a poor cosmetic result, and a recurrence. This patient has not been operated upon. The prolonged series of painful injections was of some hazard and inconvenience to these patients. The chronic draining sinus in these patients was bothersome and probably dangerous because acid-fast organisms are usually present in the discharge.

The 10 patients whose diseased glands were removed had good results. One patient had slight drainage for three weeks, which was probably attributable to the fact that a drain was used in the wound. One patient whose diseased preauricular gland was incompletely removed had drainage in small amounts for three weeks. This demonstrates the importance of complete removal of all involved glands.

Three patients had involvement of the preauricular nodes which clinically could not be distinguished from a tumor of the parotid gland. Sialograms were performed on two patients and they demonstrated the mass to be outside the parotid tissue.

In none of these patients was there any postoperative evidence of nerve damage. Streptomycin, while not always effective alone, may be of some value in the prevention of postoperative complications. In these cases streptomycin, 1 gm., was usually given daily for two weeks postoperatively. However, the authors' results with the use of streptomycin postoperatively are no better than those reported by Ladd in 1917.

Although these results were determined on a small group of patients, the authors believe they indicate that surgical incision is the best treatment for cervical tuberculous adenitis.

Streptomycin, which may fail as a lone therapeutic agent, may be of value as a postoperative adjunct to surgery,

From the Authors' Discussion and Summary.

GUMMA OF THE STOMACH IN CONGENITAL SYPHILIS. GEORGE WILLEFORD, JOHN H. CHILDERS, and W. R. HEPNER JR., Pediatrics **10**:162 (Aug.) 1952.

A gummatous lesion of the stomach due to congenital syphilis in an 11-year-old boy is described.

A history of vomiting, chronic intermittent abdominal pain, and loss of weight in a child with a positive serologic test for syphilis suggested the possibility of gastric syphilis. The roentgenologic demonstration of a constricting lesion of the stomach, gastric anacidity, and gastroscopic visualization of a red, friable, granular lesion, with ulceration, suggests a syphilitic gastric lesion. Failure to "melt" the lesion with a therapeutic trial of antisyphilitic medication prompted surgical resection of a large portion of the stomach. A lesion, compatible with the diagnosis of gumma of the stomach, was found in the gross and in the microscopic examination of the removed tissue.

FROM THE AUTHORS' SUMMARY.

RECENT EXPERIENCES WITH ANTITUBERCULOTIC ISONIAZID. G. FANCONI, Helvet. Paediat. acta **7**:540 (Dec.) 1952.

This is a short description of experiments in vivo and in vitro with isoniazid. Its action was found to be only bacteriostatic, but not bactericidal. For humans, doses of 5 to 40 mg. per kilogram a day are recommended. Because of initial reactions resembling the Herxheimer reaction, the dosage should be progressively increased during the first week, except in cases of tuberculous meningitis. In this disease cure has been reported with isoniazid alone, administered only orally. This treatment was not sufficient in two personal cases, and therefore the combination with streptomycin seems to be necessary. The most impressive results were obtained in patients already treated for meningitis tuberculosa with a poor healing tendency. The side-effects were minimal in the Swiss observations (dizziness, exanthems, leucopenia etc.); only in 0.8% of the cases the administration of the drug had to be stopped.

FROM THE AUTHOR'S SUMMARY.

ISONIAZID IN THE TREATMENT OF MILIARY AND MENINGEAL TUBERCULOSIS IN THE CHILD. C. CHOREMIS, K. PADIATELLIS, N. PRIFTIS, J. ALAMANIS and C. DANELATOU, Helvet. paediat. acta **7**:555 (Dec.) 1952.

Satisfactory results were obtained with isoniazid in five cases of miliary tuberculosis and 44 cases of tuberculous meningitis. Isoniazid has an inhibitive action on the formation of exudate and the number of the lumbar punctures could therefore be decreased in cases of tuberculous meningitis. The duration of therapy was reduced nearly twofold (three to four months).

FROM THE AUTHORS' SUMMARY.

CONTAGIOUS DISEASES IN CHILDREN WITH PRIMARY TUBERCULOSIS. A. ROSSINI and A. FATTORI, Clin. pediat. **34**:409 (June) 1952.

Over a period of two years, 265 cases of contagious diseases occurred among the children with primary pulmonary tuberculosis in a sanatorium in Forli, Italy. The following tabulation gives the number of cases in which the sedimentation rate was increased after the illness and the number of cases in which there was a worsening of the tuberculous process following the illness.

	Cases	Sedimentation Rate Increased	Tuberculous Process Worsened
Measles	58	27	1
Scarlet Fever	31	16	0
Pertussis	29	11	0
Mumps	60	18	2
Varicella	87	24	7

HIGGINS, Boston.

LOCAL TREATMENT OF SUPERFICIAL TUBERCULOUS LYMPHADENITIS WITH STREPTOMYCIN. G. MAINI, Lattante **23**:210 (April) 1952.

Twelve children with tuberculous adenitis, cervical or inguinal, were treated by injection of streptomycin locally. Better results are claimed for this method than in muscular injection of streptomycin. In cases in which the gland had caseated, the pus was evacuated by aspiration before the injection; in many of these cases, the abscess cavity was then irrigated with a 3.8% solution of sodium citrate; the rationale of the irrigation was based on the finding that tuberculous pus is acid, while streptomycin is more effective in an alkaline medium. The dose of streptomycin given was 50 to 150 mg. diluted 1 : 5 in 3.8% sodium citrate every three days.

HIGGINS, Boston

DIAGNOSIS OF SUBARACHNOID BLOCK IN CASES OF TUBERCULOUS LEPTOMENINGITIS, TREATED WITH STREPTOMYCIN. P. DURAND and A. DE MAESTRI, Minerva pediat. **4**:339 (May 15) 1952.

Hydrocephalus in tuberculous meningitis may appear (1) early owing to the increased production of spinal fluid, which is not adequately absorbed, or (2) later, owing to scar tissue formation in the meninges and at the foramina. X-ray pictures of the skull are shown to illustrate the various types of hydrocephalus and subarachnoid block found in cases of tuberculous meningitis under treatment with streptomycin.

HIGGINS, Boston.

STATUS OF THE OFFSPRING OF THE MOTHER WHO HAS RECEIVED ANTISYPHILITIC TREATMENT IN THE FORM OF PENICILLIN. L. AURICCHIO and F. VECCHIO, Pediatria **60**:261, 1952.

A follow-up study was made when the infants of 170 mothers who had been treated for syphilis with penicillin during the antepartum period were 3 months old. Fourteen of the infants had positive serum reactions. Of the other 156 infants, 68 were clinically normal, while 78 showed clinical signs suggestive of syphilis, in spite of their having a negative serum reaction.

HIGGINS, Boston.

PRIMARY TUBERCULOUS PLEURISY. G. LENCI and A. LEONETTI, Pediatria **60**:342, 1952.

The 40 cases of tuberculous pleurisy are divided into three groups following Monaldi's classification. In 33 cases, the pleurisy was due to an allergic-exudative manifestation, in two to a primary tuberculous involvement of the pleura, and in five to a caseous involvement of the lung. Thoracentesis or streptomycin was found to be of no therapeutic value against pleurisy with effusion.

HIGGINS, Boston.

CHANGES IN THE ELECTROENCEPHALOGRAM IN CHILDREN WITH TUBERCULOUS MENINGITIS BEFORE AND AFTER STREPTOMYCIN THERAPY. R. GARSCHE and G. DLUGOSCH, Ztschr. Kinderh. **69**:387 (No. 5) 1951.

Definite changes in the electroencephalographic tracings in cases of untreated tuberculous meningitis were observed. These changes improved with successful treatment. Although the changes indicate disturbances of cerebral activity, no localization was possible and the character of the changes indicated only general toxic damage by the tubercle bacilli, which might be inhibited by streptomycin.

GLASER, Jerusalem.

ANIMAL EXPERIMENTS ON THE TREATMENT OF TOXOPLASMOSIS. H. J. WOLFSCHLAG, Ztschr. Kinderh. **69**:564, 1951.

Experiments with sulfathiazole and sulfadiazine on eggs infected with Toxoplasma were without results. However, 25% of mice treated with sulfathiazole and 75% of those treated with sulfadiazine survived many weeks of observation. Antibody tests became negative and normal noninfected young were born to all the females except one mouse which remained seropositive after sulfadiazine therapy and from which Toxoplasma could be transmitted to other mice.

GLASER, Jerusalem.

Diseases of Blood, Heart and Blood Vessels, and Spleen

PROBLEMS OF HOMOLOGOUS SERUM JAUNDICE AFTER PLASMA TRANSFUSION. J. GARROTT ALLEN, A. M. A. Arch. Surg. **64**:1 (Jan.) 1952.

The incidence of homologous serum jaundice after pooled plasma transfusion is sufficiently high (5 to 15%) that many have abandoned its use save in emergencies. The author believes this is regrettable. Ultraviolet irradiation has not succeeded in "sterilizing" plasma. The experience of the University of Chicago Blood Bank is reported. No homologous serum jaundice has followed plasma transfusion during the past nine years. Storage of liquid plasma for three months at room temperature is believed to account for this experience. Suggestions are made for validating and applying this hypothesis.

YANKAUER, Rochester, N. Y.

LEVOCARDIA. H. L. MOSCOVITZ, A. J. GORDON, and S. SCHERLIS, Am. Heart J. **44**:184 (Aug.) 1952.

The authors report a case of left-sided heart (levocardia) in the presence of situs inversus of the viscera. Even though the patient was studied by both cardiac catheterization and angiocardiography, a complete anatomical diagnosis was not made. The postmortem examination revealed levocardia with situs inversus, corrected transposition of the great vessels, single ventricle, interatrial septal defects, pulmonic stenosis, and anomalous distribution of coronary arteries.

GIBSON, Chicago.

SOME OBSERVATIONS ON THE SURGICAL TREATMENT OF CONGENITAL HEART DISEASE. WILLIAM W. L. GLENN and RUTH WHITTEMORE, Ann. Surg. **135**: 297 (March) 1952.

A total consecutive series of 100 operations on 96 patients with congenital malformations of the heart and the great vessels are reviewed. There were five deaths; two of these were of patients with transposition of the great vessels.

In the course of these operative procedures, several techniques have been developed which have been of help in the management of these cases. The authors describe these techniques.

BANTIN, Omaha.

LEUKEMIA: DURATION OF LIFE IN CHILDREN TREATED WITH CORTICOTROPIN AND CORTISONE. H. R. BIERMAN, K. H. KELLY, N. L. PETRAKIS, and M. B. SHIMKIN, California Med. **77**:238 (Oct.) 1952.

The average duration of survival of 15 patients with childhood leukemia treated with corticotropin and cortisone was 6.8 months. This survival was the same as that observed among 59 children who received no treatment, or treatment with x-rays or blood transfusion alone. Despite the fact that objective evidence of remission was observed in 7 of 15 children treated with corticotropin and cortisone, the remissions were not reflected by a longer duration of life.

Treatment of childhood leukemia with corticotropin and cortisone appears to be a palliative measure, without significant effect on the duration of life.

PRYOR, Redwood City, Calif.

HEMANGIOMAS: HISTOLOGIC STRUCTURE, AND TREATMENT. L. H. WINER, California Med. **77**:242 (Oct.) 1952.

Hemangiomas are considered tumors originating in blood vessels and are classified as ectatic and hyperplastic. Controversy regarding active therapy occurs in the case of strawberry hemangiomas, which are hyperplastic capillary hemangiomas. Although they may clear up spontaneously, on rare occasions they may predispose to grave sequelae. Port-wine-stain hemangiomas (nevus flammeus) should never be treated with x-rays or radium.

PRYOR, Redwood City, Calif.

STUDIES ON ABNORMAL HEMOGLOBINS: IV. PERSISTENCE OF FETAL HEMOGLOBIN IN THE ERYTHROCYTES OF NORMAL CHILDREN. AMOZ I. CHERNOFF and KARL SINGER, Pediatrics **9**:469 (April) 1952.

Two hundred specimens of hemoglobin, prepared from the blood of children up to the age of 6 years, were investigated for their content of fetal hemoglobin. Fetal hemoglobin was usually absent after the age of 30 months, although an occasional hematologically normal subject was found to have minimal quantities of this pigment beyond this time.

The discrepancy between the results of the present investigation and data in the literature seems to be explained on the basis of the different methods used in identifying the embryonic pigment.

FROM THE AUTHORS' SUMMARY.

GLYCOGEN DISEASE OF THE HEART. ALFRED W. CHILDS, ROBERT F. CROSE, and PATRICIA H. HENDERSON, Pediatrics **10**:208 (Aug.) 1952.

Two cases of glycogen storage disease of the heart in siblings are reported.

The clinical picture was characterized by progressive weakness, areflexia, and susceptibility to secondary infections. There was also progressive hepatomegaly and cardiomegaly.

The pathologic findings correlated well with the clinical and were characterized by marked glycogen storage in skeletal, heart, and smooth muscle, in the liver, and in nerve cells of the central and autonomic nervous systems. In addition, many other tissues showed some glycogen storage. Storage in the skeletal muscle was so marked as to result in degeneration of the muscle fibers.

The familial aspect of this disease is again noted. Suggestions regarding differential diagnosis are made.

FROM THE AUTHORS' SUMMARY.

NEUROTIC MANIFESTATIONS OF INFECTIOUS MONONUCLEOSIS IN CHILDHOOD. FELIX E. KARPINSKI JR., Pediatrics **10**:265 (Sept.) 1952.

Four cases of infectious mononucleosis with central nervous system involvement are presented. The literature pertaining to the neurologic complications in infectious mononucleosis is briefly reviewed. Recovery is usually relatively prompt and complete, but permanent residual manifestations may occur.

Careful peripheral blood studies and a test for the heterophile agglutinins are essential in the etiologic diagnosis of the obscure diseases of the nervous system.

FROM THE AUTHOR'S SUMMARY.

HYPOPLASTIC ANEMIA WITH MULTIPLE CONGENITAL DEFECTS (FANCONI SYNDROME). HANS W. KUNZ, Pediatrics **10**:286 (Sept.) 1952.

A case of Fanconi's syndrome in an unusually young patient, presenting anemia, leucopenia, thrombocytopenia, microcephaly, hypogenitalism, skin pigmentation, and anomalies of thumbs, kidneys, and heart, is described. The patient's younger brother, with multiple anomalies of the extremities, hydrocephalus, abnormal ears, and abnormal genitalia died at the age of 17 months without pigmentation and hematologic symptoms, changes which usually do not occur before the age of 5 years.

The literature is reviewed and a follow-up study on previously reported cases is given. It seems that in addition to a hypoplasia of the bone marrow an extracorpuscular hemolytic mechanism plays a role in the pathogenesis of the anemia. It is suggested that some manifestations may be due to impaired adrenocortical function.

FROM THE AUTHOR'S SUMMARY AND CONCLUSIONS.

TWO CASES OF ACUTE LEUKEMIA IN CHILDREN, WITH REMISSIONS. G. MAINI and F. BARVITIUS, Lattante **20**:542 (Sept.) 1949.

Two children, aged 2 and 3½ years, with acute lymphatic leukemia, showed remissions lasting 40 and 55 days, respectively, in which the blood picture became essentially normal. The treatment preceding the remisisons consisted of injections of 480,000 units of penicillin daily and

exchange transfusions every other day, in which 150 cc. of blood was withdrawn from the patient and 300 cc. of blood of a group-O donor was injected. After the remissions, the clinical and blood pictures rapidly changed for the worse and did not respond to similar treatment.

HIGGINS, Boston.

COR TRILOCULARE BIATRIATUM. R. BULGARELLI, Minerva pediat. **3**:582 (Nov. 1) 1951.

An angiocardiographic study is included in a case report of cor triloculare biatriatum in a child of 4½ years.

HIGGINS, Boston.

BANTI'S SYNDROME IN CHILDREN AND THE VALUE OF SPLENECTOMY IN VIEW OF NEW SURGICAL VASCULAR TECHNIQUES. G. STRAMIGNONI, Minerva pediat. **3**:605 (Nov. 1) 1951.

Splenectomy was done on a 10-year-old boy with Banti's disease. The boy is in good clinical condition two years later; he has had no hemorrhages. The value of vascular shunts (splenic vein to renal vein) to lower the portal blood pressure is discussed.

HIGGINS, Boston.

HISTOCHEMICAL STUDY OF THE CELLS OF ACUTE LEUKEMIA IN CHILDREN. M. NEGRI, and L. PECCHIAI, Minerva pediat. **3**:671 (Dec. 1) 1951.

Bone marrow smears from 11 children with acute leukemia were studied with (1) Sudan black B, (2) Unna-Pappenheim and (3) May-Grünwald-Giemsa stains and (4) the peroxidase (Goodpasture's stain) and the McManus-Hotchkiss-Schiff reactions. The bone marrow leucoblasts gave negative tests for granules, fat, and peroxidases. The Schiff reaction suggested the presence of polysaccharides. The leucoblasts show such a deviation from normal in their metabolism that any attempt to classify the acute leukemias of children is both impossible and useless.

HIGGINS, Boston.

TREATMENT OF PURPURA HEMORRHAGICA (WERLHOF'S DISEASE) WITH ACTH: REPORT OF FOUR CASES. E. GRASSO and P. MENGHI, Minerva pediat. **4**:904 (Nov. 15) 1952.

Four children with purpura hemorrhagica were treated with corticotropin (ACTH) over a period of 6 to 12 days. Two of the children with purpura of three months' duration had an immediate remission, the platelet count and the clot retraction becoming and remaining normal. The third child had a similar remission, but on examination six months later the platelet count was low and the clot retraction poor; the child had had no recurrence of purpura. The fourth child was relieved of the purpura, but the increase in the platelet count and the improvement in clot retraction were apparent for only a few days. The authors recommend that in cases of purpura hemorrhagica corticotropin therapy be tried before splenectomy is done.

HIGGINS, Boston.

ACTION OF COBALT SALTS ON HEMOPOIESIS. S. VIRDIS, Pediatria **60**:302 (May) 1952.

A dose of 20 mg. of cobalt chloride by mouth, or 1 mg. cobalt in an organic preparation intramuscularly, was given to nine small children with nutritional or postinfectious hypochromic anemia daily for about 20 days. Only one child showed any signs of intolerance to the medication. Three of the children showed an increase in the red blood cell count, and five, an increase in the percentage of reticulocytes in the blood. In order to obtain an increase in the blood hemoglobin, it was necessary to give iron also.

HIGGINS, Boston.

THERAPY OF THROMBOCYTOPENIC PURPURA (WERLHOF'S DISEASE). LINNEWEH, Monatsschr. Kinderh. **97**:162 (July) 1949.

The author observed 30 cases of thrombocytopenic purpura in children from 2½ to 10 years of age. Three of the patients died. Splenectomy was performed in five cases, without mortality, and all patients have remained without symptoms and signs of the disease following surgery

The tendency toward bleeding stopped in all cases. Some patients showed an abnormally high platelet count postoperatively, but in all cases there eventually occurred stabilization of the platelet count to normal levels or to values lower than normal but higher than before splenectomy. Indications for splenectomy were confirmed diagnosis of idiopathic thrombocytopenic purpura and conservative treatment for two months with blood transfusions and administration of vitamin K and P, which also served to bring the patient into better condition for the operation. The number of thrombocytes and the presence or absence of a palpable spleen were not considered decisive factors for or against splenectomy.

HARTENSTEIN, Chicago.

STUDY OF THE DYNAMICS OF THE HEART IN THE HEALTHY CHILD. TH. HOCKERTS, Ztschr. Kinderh. **69**:431 (No. 5) 1951.

The action of the heart can be measured by three components: pressure, volume, and time. The author wanted to investigate the time factor by establishing the normal values for the *Anspannungszeit* (the time from the beginning of the systole to the opening of the semilunar valves), and the *Austreibungszeit* (the period from the opening to the closing of the semilunar valves). Carotid pulse curve and electrocardiogram and carotid pulse curve and phonocardiogram were registered together.

The first period (*Anspannungszeit*) was measured from the Q wave to the steep rise of the pulse curve minus the difference of the registration of the second tone and the aortic notch of the pulse curve. The second period (*Austreibungszeit*) was measured from the steep rise of the pulse curve to the lowest point of the aortic notch. The average values and the range for these periods at various age groups, as well as the quotient of period 1 to period 2, are reported in table form. No significant age differences could be noted.

GLASER, Jerusalem.

NEW METHOD OF EXAMINATION OF CONGENITAL MALFORMATIONS OF THE HEART. J. LEQUIME, Acta paediat. belg. **5**:80, 1951.

The second article of this series describes in some detail angiocardiography, catheterization of the heart, oxymetry, and determination of the oxygen consumption coefficient in the course of physical exercise.

GLASER, Jerusalem.

Diseases of Nose, Throat, and Ear

SPEECH DEVELOPMENT IN THE YOUNG CHILD: 1. THE AUTISM THEORY OF SPEECH DEVELOPMENT AND SOME CLINICAL APPLICATIONS. O. H. MOWRER, J. Speech & Hearing Disorders **17**:263 (Sept.) 1952.

When birds are taught to talk, it is necessary to develop a detachment of the bird from its species and to associate the vocalizations of human speech and song with pleasant experiences. The birds then develop a self-satisfaction basis of performance because of the subjective comfort provided by these sounds. Psychoanalytically and psychologically the learning of human babies may be dependent to a considerable extent on the reassuring and secondary reinforcement properties of a mother's voice, particularly as it is associated with agreeable experiences. A number of applications to speech pathology are suggested.

PALMER, Wichita, Kan.

SPEECH DEVELOPMENT IN THE YOUNG CHILD: 2. SOME FACTORS RELATED TO THE SPEECH DEVELOPMENT OF THE INFANT AND YOUNG CHILD. ORVIS C. IRWIN, J. Speech & Hearing Disorders **17**:269 (Sept.) 1952.

Studies of frequency and type of phoneme distribution in normal subjects permit studies of the influence of age, sex, race, siblings, feeblemindedness, intelligence, occupation, and similar items on variations from the normal profile. It is a reasonable inference that the development of phoneme type and phoneme frequencies presents an orderly picture. Research of this type should ultimately throw additional light on the laws of speech development of both normal and handicapped children.

PALMER, Wichita, Kan.

CLINICAL AND EXPERIMENTAL USE OF THE IOWA SCALE OF SEVERITY OF STUTTERING. DOROTHY SHERMAN, J. Speech & Hearing Disorders **17**:316 (Sept.) 1952.

The Iowa scale of severity of stuttering was employed to train observers to recognize levels of severity of the local characteristics of stuttering. Mean scale values for levels of severity were obtained for 12 five-minute samples of stuttering. Consecutive judgments were made at 9-second intervals on each sample by 11 trained observers. This method is useful for assigning rank order positions of severity to samples of uninterruptedly stuttered speech. Further experimentation is needed to demonstrate whether or not the method is useful to obtain precise scale values.

PALMER, Wichita, Kan.

A NEW HOME TREATMENT FOR EARLY CASES OF "CROUP." R. J. MEHR, New York J. Med. **53**:568 (March 1) 1953.

Acute infections of the larynx, trachea, and cronchi are seen more frequently in homes than in hospitals. The child who has been sleeping quietly suddenly awakens with a brassy cough, stridor, hoarseness, and dyspnea. His temperature is seldom much above normal in the early stages, but he is frightened, cries fearfully, and instils great dread in those about him. The condition is seldom fatal, but asphyxia, of course, could occur. The next day may be uneventful, but recurrence takes place at night, and the cycle repeats from three to five days. Mehr gives 450,000 units of penicillin with 10 mg. of diphenhydramine (Benadryl) hydrochloride (by hypodermic injection); chloramphenicol (capsules of suspension), 50 mg. per kilogram of body weight, is given without fail every six hours around the clock by the attendant; awakening the child is necessary. Because of unfavorable reports of the latter drug on the blood, Mehr has substituted oxytetracycline (Terramycin) or aureomycin with equally good results: (1) Laryngeal stridor is reduced miraculously (often within five minutes); (2) diphenhydramine calms the child and induces sleep; (3) penicillin works at once to hold back the infection until the natural resources of the body overcome the invader, and (4) the chloramphenicol, oxytetracycline, or aureomycin carries on after penicillin has tapered in value. The report is based on eight cases with no untoward effects.

ABSTRACTER'S COMMENT: This is a simple, easy method of managing an emergency, reducing the fear of parents and child, and possibly avoiding an urgent tracheotomy in certain cases.

I. W. VOORHEES, New York [A. M. A. Arch. Otolaryng.].

CHRONIC MAXILLARY SINUSITIS IN CHILDREN. J. F. BIRRELL, Arch. Dis. Childhood **27**:1 (Feb.) 1952.

The results of 382 proof punctures in 240 cases, clinically and roentgenologically diagnosed as chronic sinusitis, were positive in 13.87%, but two children (0.53%) were considered to have true chronic maxillary sinusitis. An examination of the literature in conjunction with these results leads one to the conclusion that the condition cannot be diagnosed with accuracy by any means now available. It is felt that the generally accepted theory that sinusitis is common in children is false and does not withstand critical examination. If the problem is approached with an unbiased mind it will be appreciated that identical clinical and roentgenological findings may be produced by habitual mouth-breathing. Treatment should be directed in the first instance toward reestablishment of nasal respiration rather than against a nonexistent sinus infection.

KELLY, Milwaukee.

PATHOGENESIS OF OCCULT RETROTYMPANIC OTITIS IN INFANTS. S. RAUCH, Helvet. paediat. acta **6**:528 (Dec.) 1951.

Tests on guinea pigs have shown that repeated application of 0.2 cc. of a specific antiserum in the middle ear every two to three days, beginning from birth, has no harmful effect either on the development of the middle ear or on the general condition; the parallel experiment with a mixed culture of streptococci, staphylococci, enterococci, and coli showed the same result. When, however, the antiserum and the mixed culture were applied together, both the development of the middle-ear cavity and the general development were disturbed; conditions arose which resembled occult infantile retrotympanic otitis. It is suggested that clinical investigations

be made in order to prove whether these infants actually have a predisposition for hypersensitive reactions or whether such a predisposition appears in their family history. Investigations of this sort might lead to a pathogenetic explanation of why mastoiditis caused by the same agent will result in a general toxicosis in one infant while another is unaffected.

From the Author's Summary.

Primary Tuberculosis of the Ear. J. P. DeReynier, Pract. oto-rhino-laryng. **15**:1, 1953.

The author reviews the pathogenesis and diagnosis of tuberculosis of the ear. He believes that because of the influence of the antibiotics there are many atypical cases of mastoiditis due to nonspecific infections, and these disorders are difficult to distinguish, in both clinical course and pathologic findings, from definite specific infections. He advises that all granulations and secretions from the mastoid cavity be examined histologically and bacteriologically in order to establish the exact diagnosis. On the other hand, he believes that the Koch bacillus reaches the middle ear by way of the lymphatic channels of the Eustachian tube from some localized primary focus in the pharyngeal region. Hence any tuberculous involvement of the middle ear or mastoid is a secondary phenomenon arising from some active, primary pharyngeal focus. He terms these cases primosecondary middle ear tuberculosis.

Presky, Philadelphia [A. M. A. Arch. Otolaryng.].

Malignant Epithelial Blastomata of the Nasopharynx in Children. M. Orlandelli, Clin. pediat. **34**:377 (June) 1952.

Two cases are reported, those of an 11-year-old girl and a 10-year-old boy, who had been admitted to the hospital with diagnoses of tuberculous cervical lymphadenitis. The finding of negative tuberculin reactions was followed by thorough nasopharyngeal examinations, which revealed a tumor, the size of a cherry, in the posterior nasopharynx of the girl, and a small polypoid mass near the opening of a frontal sinus of the boy. The histological diagnosis in each case was malignant epithelial blastoma. While such malignant growths are rare in children, careful examination of the nasopharynx is important in cases of nontuberculous cervical adenitis.

Higgins, Boston.

The Treatment of Acute Laryngitis in the Infant with the Antihistaminics. Rafael de la Portilla, Rev. cubana oto-laryng. **111**:41 (Jan.) 1953.

The author discusses the problem of acute laryngitis in infants and young children. He presents a typical picture of the disease with all its serious complications. In reviewing the various therapeutic measures he believes that many of these patients can be benefited by the intensive use of the antihistaminics. This is particularly true in the case of laryngitis which is not due to diphtheria. He uses a number of antihistaminic drugs. For the child of 2 to 4 years of age he gives 50 mg. of diphenhydramine (Benadryl) tripelennamine (Pyribenzamine) every four hours for 2 doses; then he gives 25 mg. every four hours until there is a complete improvement in the symptomatology. For children 6 months to 2 years of age he gives 25 mg. every four hours for 2 doses and then 25 mg. every six hours. He has not had occasion to continue the medication for more than 48 hours. For a few patients he had to supplement the treatment with the antibiotics and vapor therapy, particularly when there were signs of an acute bronchial infection. He has observed that (1) this form of therapy has produced a rapid disappearance of obstructive symptoms and converted the entire picture into a simple tracheobronchitis, which was easily treated by steam inhalation; (2) there were no late complications, such as pulmonary atelectasis and pneumonic foci; (3) the time of hospitalization was markedly reduced (all his patients left the hospital within two days, except one patient who remained for three days), and (4) there was not a single instance in which surgical intervention was necessary.

Presky, Philadelphia [A. M. A. Arch. Otolaryng.].

PROBLEMS OF DIAGNOSIS OF TONSILLAR FOCI. WALTER BECKER, Deutsche med. Wchnschr. **76**:1072 (Aug. 31) 1951.

Tests with material obtained by puncture of the tonsils advocated by Schulz and Leiber were made. A positive skin reaction with consequent increase of eosinophiles after 48 to 72 hours is assumed to indicate a focus in the tonsils as well as its activity. These claims could not be fully substantiated. Details of this work will be published elsewhere.

AMBERG, Rochester, Minn.

NEW CURATIVE PROCEDURE FOR MIDDLE EAR DISEASES. L. FALTA, Deutsche med. Wchnschr. **77**:82 (Jan. 18) 1952.

In cases of beginning catarrhal and acute middle ear infections irrigations of the auditory canal with water heated to 45 C. are recommended. The irrigations, by means of a 150 ml. syringe, are repeated three times and may be repeated on successive days. Pain is relieved quickly. It is claimed that the necessity for paracentesis of the drum membrane may be obviated frequently. In the presence of a perforation or imminent perforation the procedure is contraindicated. Children below the age of 7 or 8 years require some assistance.

AMBERG, Rochester, Minn.

Diseases of Lungs, Pleura, and Mediastinum

MEDIASTINAL GASTRIC CYST: SUCCESSFUL EXCISION IN AN EIGHT-WEEK-OLD INFANT. MARK W. WILLIAMS and JAMES F. JOHNSON, A. M. A. Arch. Surg. **64**:138 (Feb.) 1952.

Successful excision of a mediastinal gastric cyst in an 8-week-old infant is reported, together with a review of 32 published cases. This condition is invariably symptomatic in infancy, and it may present with urgent indications for operation. It may be suspected when the chest x-ray indicates a rounded posterior mediastinal mass.

YANKAUER, Rochester, N. Y.

PRESSURE-TIME RELATIONS IN THE SAFE CORRECTION OF ATELECTASIS IN ANIMAL LUNGS. RICHARD DAY, ALICE M. GOODFELLOW, VIRGINIA APGAR, and GUSTAV J. BECK, Pediatrics **10**:593 (Nov.) 1952.

Observations on animal lungs support the following statements: 1. Injury to lungs is caused by overdistention. 2. Distention is related to time as well as to the pressure exerted. 3. A certain minimum pressure must be exerted to correct atelectasis; to employ this pressure safely, its duration must be limited.

The combinations of pressure and time which produce satisfactory inflation in rat, rabbit, and pig lungs are very similar to one another. Research is needed to find the combinations which are utilized by the normal newborn infant and which might be suitable in positive pressure resuscitation.

No practical application of the methods of inflation which have been described has so far been attempted. Small differences in the duration of each positive pressure inflation determine the safety of the procedure. Clinical application is a subject for future research.

FROM THE AUTHORS' SUMMARY.

DEXTROCARDIA SECONDARY TO FOREIGN BODY IN LEFT BRONCHUS: REPORT OF TWO CASES. MURDOCK EQUEN, FRANK BUCKNER, GEORGE ROACH, and ROBERT BROWN, South. M. J. **45**:16 (Jan.) 1952.

The authors report on two infants who had aspirated foreign bodies, the presence of which was not recognized for 4 weeks in one instance and 10 weeks in the other. Each infant became markedly dyspneic when seen by the reporting physician. The mediastinal structures and the heart were pushed to the right by a marked emphysema of the left lung. Both infants recovered after removal of the foreign body: a piece of bone in the first child and a sandbur in the second.

LOGAN, Rochester, Minn.

OIL ASPIRATION PNEUMONIA IN INFANCY. F. S. W. BRIMBLECOMBE, L. CROME, and J. P. M. TIZARD, Arch. Dis. Childhood **26**:148 (April) 1951.

The history of the recognition of oil aspiration pneumonia (lipid pneumonia) is reviewed. A case of fatal paraffin pneumonia in an infant of 8½ months is described from the clinical and pathological standpoints. The etiology of the condition in infants and its prevention are discussed.

KELLY, Milwaukee.

PNEUMONIA AND EMPYEMA IN CHILDREN. DUNCAN MACAULAY, Arch. Dis. Childhood **27**:111 (Feb.) 1952.

Data about the patients with acute respiratory infection admitted to the Royal Edinburgh Hospital for Sick Children from April, 1947, to March, 1949, are presented and compared with those available from earlier reports from this hospital. The type of disease encountered does not appear to have changed appreciably in the past 30 years, and fairly constant proportions of the two principal types of pneumonia at various ages are shown to have been observed. The mortality from lobar pneumonia showed a steady decline from 1908 to 1949; this decline is largely unexplained. Staphylococci have replaced pneumococci as the principal cause of acute empyema in this hospital. This change is related to, but not completely explained by, the introduction of chemotherapeutic drugs.

KELLY, Milwaukee.

CONGENITAL AIR-CONTAINING CYSTS OF THE LUNGS IN CHILDREN. A. ZANINI and L. PARENZAN, Minerva pediat. **3**:615 (Nov. 15) 1951.

Two cases of congenital cysts of the lung in children under one year of age are reported. In one child, a large cyst of the upper lobe of the left lung was one of several congenital malformations (Parkes Weber syndrome); the cyst would occasionally fill with fluid during some of many attacks of bronchopneumonia. The other infant was premature, the cyst being in the lower lobe of the left lung, behind the heart. The differential diagnosis of congenital and acquired pulmonary cysts is discussed at some length.

HIGGINS, Boston.

ARTIFICIAL ANTERIOR PNEUMOMEDIASTINUM IN CHILDREN. L. GUFFONI, Minerva pediat. **3**:631 (Nov. 15) 1951.

Condorelli's method for injecting air into the anterior mediastinum as a diagnostic aid in cases of enlarged thymus or suspected tumor is described. The procedure is reported as safe and technically not difficult.

HIGGINS, Boston.

PLASMA-CELL PNEUMONITIS IN THE INFANT. F. TECILAZICH, Minerva pediat. **3**:657 (Nov. 15) 1951.

Plasma-cell pneumonitis is a disease of infants under 5 months of age and is characterized by infiltration of plasma cells into the alveoli. The symptoms are usually dyspnea, mild cyanosis, and poor circulation. It lasts about eight days and in hospital epidemics may have a mortality rate as high as 50%. It is probably caused by a virus, although this has not been proved. The white blood count is usually elevated and the sedimentation rate increased. Antibiotics do not seem to be of any benefit, but short-wave therapy has given some symptomatic relief.

HIGGINS, Boston.

Diseases of the Gastrointestinal Tract, Liver, and Peritoneum

SURGICAL JAUNDICE IN INFANTS AND CHILDREN. EDWIN W. GERRISH and JACK W. COLE, A. M. A. Arch. Surg. **63**:529 (Oct.) 1951.

Eighty-four cases of jaundice in children under 12 years of age seen at the authors' hospital are analyzed. Forty-one cases (including cases of congenital hemolytic anemia) were "surgical." Physiological jaundice and erythroblastosis were excluded from both groups. Extrahepatic

biliary obstruction is very rare after 6 months of age. The causes of biliary obstruction in early infancy are discussed. Two cases of common duct strictures believed due to chronic pancreatitis are described in detail. These strictures occurred at 2 and 2½ years of age. The authors caution against ascribing common duct obstruction to enlarged lymph nodes.

YANKAUER, Rochester, N. Y.

PATENT OMPHALOMESENTERIC DUCT. HOWARD B. KIRTLAND, A. M. A. Arch. Surg. **63**:706 (Nov.) 1951.

The etiology, signs, and symptoms of patent omphalomesenteric duct are discussed, and a case is reported in which the complication of ileal prolapse was successfully treated surgically. This complication occurs in 20% of the cases, and only four operative recoveries have been previously reported.

YANKAUER, Rochester, N. Y.

ACUTE INTESTINAL OBSTRUCTION IN INFANCY. CHARLES W. MCLAUGHLIN JR. and JOHN D. COE, A. M. A. Arch. Surg. **64**:541 (May) 1952.

The cases of 53 consecutive infants with acute intestinal obstruction are analyzed and discussed. Diagnoses in order of frequency are intussusception, duodenal obstruction, small bowel obstruction, incarcerated or irreducible inguinal hernia, and imperforate anus. Cases of pyloric obstruction were excluded. The symptoms, age of onset, diagnosis, and treatment are discussed, and certain general principles of handling emphasized.

YANKAUER, Rochester, N. Y.

APPENDICITIS DURING THE FIRST TWO YEARS OF LIFE: REPORT ON TWENTY-ONE CASES AND REVIEW OF FOUR HUNDRED FORTY-SEVEN CASES FROM THE LITERATURE. WILLIAM H. SNYDER JR. and LAWRENCE CHAFFIN, A. M. A. Arch. Surg. **64**:549 (May) 1952.

Four hundred forty-seven cases from the literature are reviewed with a report of 21 additional cases. Etiology, symptoms, and treatment are discussed. Appendicitis is not a common acute abdominal emergency in infancy. One-seventh as many cases occur in the first year of life as in the second. In 71% of the cases reviewed, perforation had occurred before the diagnosis was made. The mortality in the literature was 29% and in this series of 21 cases 19% (all deaths occurred prior to the introduction of antibiotics). The value of supportive measures in therapy is stressed.

YANKAUER, Rochester, N. Y.

ACUTE APPENDICITIS IN INFANTS: FIFTEEN-YEAR STUDY. CLIFFORD D. BENSON, JOHN J. COURY JR., and DONALD R. HAGGE, A. M. A. Arch. Surg. **64**:561 (May) 1952.

A series of 39 infants (under 2 years) treated over a 15-year period is reported. Incidence, symptoms and diagnosis, pathology, treatment, and results of treatment are discussed. Only three cases occurred in the first year of life. Although the over-all mortality rate was 15%, there were no deaths in the final 10 years of the study. In 32 cases perforation had occurred prior to operation. The authors believe that education, increased use of surgery, and the advent of antibiotics have been responsible for the reduction in mortality.

YANKAUER, Rochester, N. Y.

DEFECTS IN RIGHT DIAPHRAGM OF INFANTS AND CHILDREN WITH HERNIATION OF LIVER. MARK M. RAVITCH and JACOB C. HANDELSMAN, A. M. A. Arch. Surg. **64**:794 (June) 1952.

Five cases are reported in which attenuation of the right diaphragmatic muscle, varying in extent from half its area to 4 cm. in diameter, occurred. In all cases the liver protruded through the weakened area, producing a syndrome analogous to diaphragmatic hernia. In three cases there were associated symptoms. In all cases surgical correction was done. The authors believe that this abnormality should be considered in the differential diagnosis of masses low in the right chest and that operation is indicated in all cases for the same reasons as it is indicated in the usual cases of diaphragmatic hernia.

YANKAUER, Rochester, N. Y.

HEMOBILIA, CHOLECYSTITIS, AND GASTROINTESTINAL BLEEDING WITH RUPTURE OF LIVER. HARRY J. EPSTEIN, and BENJAMIN LIPSHUTZ, J. A. M. A. **149**:1132 (July 19) 1952.

A 9-year-old boy sustained a traumatic rupture of the liver. The wound was sutured; however, the liver continued to bleed into the biliary passages and distended the gall bladder to about eight times its normal size. This was surgically drained, and the boy recovered from both operations. A few similar cases have been reported before.

KIMBALL, Evanston, Ill.

THUMB-SUCKING AND THE ORAL STRUCTURES: A SERIAL STUDY FROM BIRTH TO 14 YEARS OF AGE. J. H. SILLMAN, J. Pediat. **39**:424 (Oct.) 1951.

Thumb-sucking in children under the age of 4 years does not cause buck-teeth. In older children with malocclusion, it may accentuate the deformity.

MACDONALD, Pittsburgh.

USE OF TRANSPARENT TAPE IN THE TREATMENT OF UMBILICAL HERNIA IN INFANTS. GEORGE E. PRINCE, J. Pediat. **39**:481 (Oct.) 1951.

Cellophane tape is less irritating than ordinary adhesive in the strapping of umbilical hernias in infants.

MACDONALD, Pittsburgh.

UMBILICAL HERNIA: I. OCCURRENCE OF THE INFANTILE TYPE IN NEGRO INFANTS AND CHILDREN. E. PERRY CRUMP, J. Pediat. **40**:214 (Feb.) 1952.

Although umbilical hernia is apparently about eight times as frequent in the Negro as in the white person, Crump was unable in this study to implicate either sex or prematurity as causative factors. The condition is benign; only 1 of these 1,237 infants required surgical treatment.

MACDONALD, Pittsburgh.

OMENTAL SEGMENTAL INFARCTION: A REPORT OF TWO CASES IN CHILDREN. FRANCIS P. CATANZARO and JOHN E. FARLEY JR., J. Pediat. **40**:240 (Feb.) 1952.

Catanzaro and Farley report two cases of omental segmental infarction, a condition usually associated with vascular disease in adults and rarely seen in children. The patients reported here are a 10-year-old white boy and a 4-year-old white girl. Both patients were moderately obese. The symptoms simulated appendicitis. In both cases, the right lower edge of the omentum was involved. This is the usual area affected in these cases. Excision of the infarcted segment resulted in uncomplicated convalescence.

MACDONALD, Pittsburgh.

A COMPLETE CLEFT PALATE PROGRAM. WAYNE B. SLAUGHTER and GRETCHEN MUELLER PHAIR, J. Speech & Hearing Disorders **17**:123 (June) 1952.

For the past 17 years in the State of Wisconsin about 1 in every 770 infants born has had a cleft lip or palate which must be reported by the attending physician. Diagnosis is made at the University of Wisconsin Medical School. A social service interview is included in the medical examination. After surgery, a formal training of eight weeks is given these children, including hearing tests, psychometric studies, reexamination of the palate, etc. Satisfactory rehabilitation is a long-time program and must be continued until the child is a normal member of society.

PALMER, Wichita, Kan.

FAT DISEASE OF THE LIVER IN INFANTS ON THE ISTHMUS OF PANAMA. FRED H. HOWARD and WILLIAM A. MERIWETHER, Pediatrics **10**:150 (Aug.) 1952.

Forty-eight cases of fatty liver disease in infancy coming to autopsy over the past 10 years at Gorgas Hospital are reported. The diagnosis of this condition is extremely difficult clinically; liver biopsy is the only certain way of making the antemortem diagnosis. In Panama, vomiting, diarrhea, and fever are the most frequent presenting complaints. Physical findings of help are the stunted growth, fever, and possibly enlarged liver. Other findings seen commonly in the African cases, such as edema, glossitis, and dermatosis, are usually not seen. The essential pathologic finding is a markedly fatty liver. Pancreatic changes consisting of acinar atrophy

were seen in some cases. The cause of the deficiency state appears to be an inadequate protein intake, and it will not be simple to deal with, as poverty and ignorance, rather than neglect, would seem to be responsible for this disease. The prognosis is extremely poor, only 11 of the 48 patients surviving for more than 24 hours after medical attention was sought. It is probable that fatty liver disease in infants is relatively common in Panama but almost completely overlooked clinically.

FROM THE AUTHORS' SUMMARY.

COMBINED PELVIC AND RETROPERITONEAL TERATOMAS IN INFANCY AND CHILDHOOD. ERNEST E. ARNHEIM, Pediatrics **10**:198 (Aug.) 1952.

The pathologic and clinical features, diagnosis, and treatment of combined pelvic and retroperitoneal teratomas in infancy and childhood, based upon a study of 15 cases, are reviewed. One case of malignant pelvic and retroperitoneal teratoma with recovery for four years eight months is presented; this is believed to be the first reported cure of this condition.

Combined pelvic and retroperitoneal teratomas were usually noted in female infants under the age of one year as large tumors in the pelvis and lower abdomen, with or without extension into the buttocks, often causing urinary or intestinal obstruction, readily diagnosed by rectal and abdominal examination, and revealing areas of calcification or ossification on roentgenologic examination.

In 4 (38%) of this group of 15 cases, the teratomas had undergone malignant changes, and in 4 operation had not been done and the teratomas were described at autopsy.

Successful treatment of combined pelvic and retroperitoneal teratomas was dependent upon early operation, careful operative technique through staged posterior and abdominal incisions, and adequate preoperative and postoperative management. The operative mortality was 20%.

A long-term recovery rate of 15% for combined pelvic and retroperitoneal teratomas is in marked contrast to the reported cures for sacrococcygeal teratomas (68%) and retroperitoneal teratomas (42%).

FROM THE AUTHOR'S SUMMARY.

PROLONGED OBSTRUCTIVE JAUNDICE IN INFANCY: 1. GENERAL SURVEY OF 156 CASES. DAVID YI-YUNG HSIA, PAUL PATTERSON, FRED H. ALLEN JR., LOUIS K. DIAMOND, and SYDNEY S. GELLIS, Pediatrics **10**:243 (Sept.) 1952.

One hundred fifty-six cases of prolonged obstructive jaundice seen at the Children's Medical Center between 1940 and 1951 have been investigated and in 85% of the cases follow-up studies have been carried out. In 94 (61%) the jaundice was due to true biliary atresia. In 23 (15%) it was due to inspissated bile associated with erythroblastosis. All the patients are living and well and free of jaundice. The diagnosis of this condition can be made when the patient has (1) jaundice during the first two days of life with persistence of jaundice for over three weeks, (2) high values for direct and indirect reacting bilirubin compatible with obstructive jaundice, (3) negative flocculation tests, and (4) blood-grouping studies consistent with erythroblastosis.

Thirty patients (19%) had jaundice due to inspissated bile of unknown etiology. Of these, 6 are dead, 2 are alive but cirrhotic, and 18 are living and well; no follow-up was obtained on the other 4. There appears to be some relationship between this condition and immaturity of the liver, small extrahepatic ducts, thickened secretions, and possibly dehydration.

In nine (6%) of the cases the jaundice was due to miscellaneous causes, in four of which it was probably due to infectious hepatitis.

FROM THE AUTHORS' CONCLUSIONS.

STUDIES IN PANCREATIC FIBROSIS: COR PULMONALE; CLINICAL AND PATHOLOGIC OBSERVATIONS. ALEXANDER S. NADAS, GILBERT COGAN, BENJAMIN H. LANDING, and HARRY SHWACHMAN, Pediatrics **10**:319 (Sept.) 1952.

The cardiac complications of pancreatic fibrosis were studied by clinical examination and autopsy.

Of 45 patients with severe pancreatic fibrosis studied clinically, 8 showed cor pulmonale. One of these eight died in right-heart failure; the others showed only electrocardiographic changes characteristic of right ventricular hypertrophy.

A comparison of the autopsy findings of 35 patients with pancreatic fibrosis with those of a control series showed no diagnostic differences in cardiac weight or absolute right ventricular thickness. The ratio of right to left ventricular thickness, however, was higher in the group with pancreatic fibrosis, as was the ratio of cardiac weight to body weight. In only 2 of 35 autopsies were visceral edema and congestion of significant degree shown, suggestive of frank right ventricular failure.

Physiologic and pathologic considerations are presented concerning the development of pure pulmonary hypertension and its relationship to cor pulmonale, compensated or decompensated.

The data presented here suggest that the pulmonary involvement associated with infection is still the principal threat to life in patients with pancreatic fibrosis and the one toward which major therapeutic efforts should be directed.

FROM THE AUTHORS' SUMMARY.

A FAMILIAL CRISIS IN CONGENITAL ACHOLURIC JAUNDICE. W. N. INGHAM, Arch. Dis. Childhood **27**:36 (Feb.) 1952.

A familial crisis affecting five members of a family with congenital acholuric jaundice is described. Consideration of this and other reported families does not support the view that infection is the trigger mechanism concerned.

KELLY, Milwaukee.

THE TRYPTIC ACTIVITY AND PRESENCE OF CORNIFIED SQUAMES IN MECONIUM AS A DIAGNOSTIC AID IN CONGENITAL INTESTINAL OBSTRUCTION. JOHN L. EMERY, Arch. Dis. Childhood **27**:71 (Feb.) 1952.

It was found that in 92% of 142 specimens from 100 normal infants, tryptic activity was present in meconium to a dilution of at least 1 in 8. In five cases of intestinal atresia, tryptic activity was absent in four and present at a dilution of 1 in 2 in one case.

Meconium smears from 92 normal children and 4 children with congenital atresia were stained as recommended by Farber and sent to three general clinical pathologists. Their reports were consistent and correct in only 16 of the 96 cases. This was found to be largely due to staining difficulties, and a more contrasting stain showed cornified squames in all smears but varying greatly in quantity. Squames were also found in small numbers in the smears from infants with atresia. Farber's test modified in this way should be considered positive only if large numbers of cornified squames are found.

If tryptic activity is absent in meconium, atresia below the ampulla of Vater or meconium ileus is likely. Tryptic activity present at a dilution of 1 in 8 or more would appear to exclude atresia below the duodenum.

KELLY, Milwaukee.

FIBROCYSTIC DISEASE OF THE PANCREAS WITH MECONIUM PERITONITIS AT BIRTH. F. W. NASH and J. F. SMITH, Arch. Dis. Childhood **27**:73 (Feb.) 1952.

A case of meconium peritonitis in association with fibrocystic disease of the pancreas in a newborn infant is reported. Careful examination of the main pancreatic duct by serial section revealed no mechanical obstruction or stenosis. Features of particular interest were the presence of abdominal distention at birth, the absence of vomiting, which is usually a prominent feature of most cases of meconium ileus, and the unusual site of intestinal perforation in the transverse colon.

KELLY, Milwaukee.

CHYLOUS ASCITES IN INFANCY. I. KESSEL, Arch. Dis. Childhood **27**:79 (Feb.) 1952.

A case of chylous ascites occurring in a Bantu girl in the third month of life is described. Altogether 104 oz. (3,120 ml.) of this fluid was removed by four successive abdominal tappings, and in addition a further 20 oz. (600 ml.) was found in the peritoneal cavity at necropsy. On analysis the fluid was shown to resemble true chyle. The histopathologic examination suggested a congenital anomaly of the lymphatic drainage of the intestinal tract as the etiological factor.

KELLY, Milwaukee.

CONGENITAL STENOSIS OF THE ESOPHAGUS WITH LATE APPEARANCE OF SYMPTOMS. G. CHARLEUX, P. MOUNIER-KUHN, R. BRETTON, and J. DAVID, Pédiatrie **40**:539, 1951.

The cases of three children are described, 1, 2, and 2½ years of age, who showed late symptoms of esophageal obstruction.

In the discussion, the authors point out that because of the late appearance of symptoms the correct diagnosis of esophageal stenosis is rarely made, that roentgenography with barium is essential for the diagnosis, and that by esophagoscopy one can determine whether the mucosa is normal and the lesion congenital or due to injury to the mucosa. Of the congenital lesions, the simplest is a diaphragm with a small opening permitting liquid food to pass or stenosis over a shorter or longer distance with or without bronchial fistula.

In the differential diagnosis one must consider spasms, brachyesophagus, scarring of the mucosa, and foreign body. The treatment consists in resection of the diaphragm, progressive dilatations from above or below, or resection of part of the esophagus.

GLASER, Jerusalem.

TREATMENT OF DIARRHEA IN INFANTS AND CHILDREN WITH HIP MEAL AND HIP EXTRACT. G. IVADY, Ann. pædiat. **178**:107 (Feb.) 1952.

The author recommends hips for the treatment of diarrhea (he mixes one part of 2.5% gruel with one part of a 20% hip flour decoction). The result is superior to the grated apple diet, but the stools are less consistent than after carrot soup.

In order to obtain an acid milk he recommends instead of lemon juice the addition of 2 teaspoonfuls of 40% hip extract to 100 ml. of milk.

Hip flour and extract can be combined with other antidyspeptic measures. Apart from these advantages hip flour is cheap, easily prepared and can be used at any season.

AMBERG, Rochester, Minn.

LYMPHOSARCOMA WITH INVAGINATION OF THE CECUM OF AN 8-YEAR-OLD BOY; FIVE-YEAR-CURE FOLLOWING COLECTOMY. G. PLACITELLI, Clin. pediat. **34**:369 (June) 1952.

A diagnosis of recurrent appendicitis followed by appendectomy was made two and one-half months after a 9-year-old boy began to complain of right lower quadrant pain following meals. The symptoms continued, and six weeks later a tumor was felt in the right lower quadrant. At operation, a tumor the size of a small lemon was found invaginating the large intestine; a colectomy was done. The histological diagnosis was lymphoblastic sarcoma. The child was seen five years later and found to be in good health.

HIGGINS, Boston.

CONGENITAL MICROCOLON. A. GIANELLI, Minerva pediat. **4**:915 (Nov.) 1952.

Three fatal cases of microcolon in newborn infants are reported. The small sizes of the colons were not due to an obstruction of the colon itself, but were secondary to anatomical or functional obstructions higher in the intestine.

HIGGINS, Boston.

ACUTE APPENDICITIS IN INFANTS AND CHILDREN. U. Bucci, Pediatria **59**:838, 1951.

The symptoms and signs found in each of 28 cases of appendicitis in children under 8 years of age are recorded. There were two deaths. The danger of perforation in children is so great that early diagnosis is imperative; one must also be acquainted with the signs of the disease in children, since they are somewhat different from the signs in adults.

HIGGINS, Boston.

LEFT PARADUODENAL HERNIA IN AN INFANT. B. CARLETTI and G. PELLEGRINI, Pediatria **59**:846, 1951.

In an infant of 2½ months an acute intestinal obstruction developed. At autopsy it was found to be due to a paraduodenal hernia with invagination of the jejunum.

HIGGINS, Boston.

HIRSCHSPRUNG'S DISEASE AS A TYPE OF MEGACOLON. A SOSTEGNI and G. STRAMIGNONI, Pediatria **60**:308, 1952.

There are three types of megacolon: the symptomatic, the functional, and the aganglionic. The symptomatology is secondary to some organic disease and may be relieved by overcoming the primary condition. The functional, or idiopathic, type does not respond to surgical intervention. The ganglionic type, or true Hirschsprung's disease, is due to abnormality of the nerve plexuses of a section of the colon. Surgical resection of the involved part of the intestine is described, according to technique described in the American literature.

HIGGINS, Boston.

HEMORRHAGE IN PARTIAL GASTRIC ECTOPIA IN THE CHILD (DIAPHRAGMATIC HERNIA). JUAN PICANOL, Acta pediat. españ. **118**:822 (Oct.) 1952.

The author reports two cases of partial gastric ectopia in infants in which the first symptom was vomiting of hemolyzed chocolate-colored material mixed with mucus. X-rays from the two cases and full clinical descriptions are included.

The origin of the hemorrhages is believed to be the constriction of the diaphragmatic ring around the herniated stomach, resulting in venous stenosis, edema, and finally ulcerations.

Frequently melena and anemia indicate only the presence of a diaphragmatic hernia, but vomiting of old blood certainly indicates a need for surgical intervention.

ALLEN, San Francisco.

A HYDATID CYST OF THE FLOOR OF THE MOUTH. PEDRO L. ERRECART and JOSE M. CASTILLO MORALES, Acta oto-rhino-laring. Ibero-Am. **3**:318, 1952.

The authors report a case of a swelling of the floor of the mouth of a 12-year-old child, in whom the mother noted a sense of obstruction to eating and to talking for four months. Under the tongue was a tumor which increased in size without pain. The initial diagnosis was a ranula. However, on extirpation it was found to be a hydatid cyst on the floor of the mouth. The patient made an uneventful recovery.

PERSKY, Philadelphia [A. M. A. Arch. Otolaryng.].

LIVER FUNCTION TESTS IN INFECTIOUS HEPATITIS IN CHILDHOOD. G. A. v. HARNACK, Ztschr. Kinderh. **70**:10 (No. 1) 1951.

The author analyzes the value of various liver-function tests as to their diagnostic significance and their ability to indicate the course, particularly the end, of the disease. Normal and border values are quoted for all tests, and the exact techniques of the hippuric acid and the galactose tests are described. On the basis of experience in investigating some 30 children, the author describes in graph form the time after which each one of these tests becomes negative. It seems that the thymol-turbidity test remains positive the longest and is therefore the most reliable test in the establishment of recovery, although, because of its nonspecific nature, it is not diagnostic unless used in combination with other tests.

The seriousness of the hepatitis can best be determined by the hippuric acid and the galactose test.

GLASER, Jerusalem.

BACTERIOLOGY OF ENTERITIS. W. ZISCHKA, Wien. klin. Wchnschr. **64**:758 (Sept. 19) 1952.

The author discusses the relation of certain strains of Escherichia coli to enteritis of infants. Bacterium coli III B4 was grown from the feces in 70% of 108 infants. It was present in 30% of 93 contacts. These represent carriers. Nearly all these infants were infected in the hospital, and the correlation of the presence of this strain with the onset of the disease was such that finding of the bacteria preceded the symptoms by about two days. At the height of the disease it was the only micro-organism demonstrable. Bacterium coli III B4 was also found in gastric juice, in nose and throat, in urine, and in the pus of an intervening otitis media. It could be grown from various external causes of contamination.

At postmortem examination the micro-organism was found in small and large intestine, stomach content, bile, mesenteric lymph nodes, often in spleen, and in complicating pneumonias and otitis media. Kittens could be infected with infected milk. It is concluded that the majority of cases of infantile enteritis, but not all, are caused by certain types of bacteria belonging to the coli group.

AMBERG, Rochester, Minn.

AUREOMYCIN IN APHTHOUS STOMATITIS. W. RITTNER and O. THALHAMMER, Wien. klin. Wchnschr. **64**:996 (Dec. 26) 1952.

Aureomycin is applied to the mouth after a meal and after rinsing. The treatment is simple and painless. The drug is to remain in the mouth. The dose is 50 mg. three times a day, usually for two to four days. The cost of the drug is less than that of a day in the hospital, and certainly justified, taking into consideration the suffering of the children.

AMBERG, Rochester, Minn.

CONGENITAL OBSTRUCTION OF THE DUODENUM: REPORT OF FIVE SURGICALLY TREATED CASES. P. DENYS, Acta paediat. belg. **5**:7, 1951.

The author reports five cases of duodenal obstruction in three of which the obstruction was definitely extrinsic (due to compression of the intestine from without) and in two probably intrinsic (exact investigation was not possible during the operation). Contrary to some reports, Denys believes that extrinsic causes are by far more frequent than intrinsic causes and that the pathogenesis is not fetal peritonitis but rather malrotation of the intestine and obstruction due to mesenteric bands.

Since freeing of the of the duodenum may entail tears and perforation, gastrojejunostomy or duodenojejunostomy is recommended. Deny's first patient underwent four operations and reached an age of 36 days before passage of food was established. This case demonstrates the fact that newborn infants can survive malnutrition and operative shock well if treated parenterally. The fourth patient died of bronchopneumonia 12 hours after surgery.

The symptomatology of complete intestinal obstruction is reviewed, and the difficulty of diagnosing partial duodenal obstruction is pointed out. Pains may be one of the symptoms leading to the diagnosis of partial obstruction and may be recognized by the appearance of the infants' face and prolonged complaining crying.

GLASER, Jerusalem.

ATRESIA OF THE ESOPHAGUS: I. FROM THE PEDIATRICIAN'S VIEWPOINT. L. MARIS, Acta paediat. belg. **5**:21, 1951.

The author reports on five patients with esophageal atresia with tracheoesophageal fistula, of whom one died of rupture of sutures and mediastinitis, two died of postoperative pulmonary complications, and two recovered. It is pointed out that the results depend greatly on the early diagnosis and transfer of the child into the hands of the surgeon as well as on the preoperative and postoperative care.

GLASER, Jerusalem.

Nervous Diseases

PHOTOGENIC EPILEPSY IN THE CHILD. M. TURNER and H. GASTAUT, Arch. pediat. **22**:309 (Nov.-Dec.) 1951.

Rhythmic photostimulation is a very effective method to activate the electroencephalograms of known or latent epileptics. By its innocuity and by its easy application it is specially indicated in infancy.

Photogenic epilepsy in childhood has a clinical individuality very significant in neuropediatrics by its etiologic, clinical, electroencephalographic, and evolutive characteristics: It means a neuronic perturbation genetically transmitted and very sensible to the trimethiadonic treatment.

This concept and the electroencephalographic technique explain the physiopathogeny of the important group of "genetic" or "idiopathic epilepsy"; they amplify the limitation of the "petit mal" (in Gibbs's and Lennox' meaning). They agree with the Montreal School's classification of epilepsy and increase the trimethiadonic therapy.

AUTHORS' SUMMARY.

DIAGNOSIS AND TREATMENT OF MENINGITIS. ARCHIBALD L. HOYNE, Illinois M. J. **100**:285 (Nov.) 1951.

In this comprehensive review, Hoyne discusses the differential diagnostic features of the various forms of meningitis in the different age levels of infancy and childhood. The advantages of the sulfonamide drugs and antibiotics in the treatment of the various types of meningitis are pointed out.

The sole purpose of a lumbar puncture, according to Hoyne, should be diagnostic when other means are not adequate. Intrathecal treatment is contraindicated for every form of meningitis. After a bacteriologic diagnosis has been established, the cerebrospinal fluid generally requires no further thought.

Barbour, Peoria, Ill.

LANGUAGE DEVELOPMENT IN A GROUP OF CHILDREN WITH SPASTIC HEMIPLEGIA. SHULAMITH KASTEIN and JETTA HENDIN, J. Pediat. **39**:476 (Oct.) 1951.

Language development in spastic patients depends on mental potential rather than on the severity or side of impairment or handedness.

MACDONALD, Pittsburgh.

HEREDITARY PERIODIC PARALYSIS: II. EFFECTS OF FASTING AND OF VARIOUS TYPES OF DIET ON OCCURRENCE OF PARALYTIC ATTACKS. I. McQUARRIE and M. R. ZIEGLER, Metabolism **1**:129 (March) 1952.

The relationship between diet and incidence of paralytic attacks was studied in a 15-year-old boy suffering from familial periodic paralysis. Spontaneous episodes of hypopotassemia and paralysis were found to be abolished by fasting. Since there is a well-established relationship between excessive ingestion of carbohydrate and occurrence of attacks, quantitative measurements were made of the ratios of amounts of carbohydrate to potassium ingestion of which would lead to episodes of paralysis. Whenever the ratio of carbohydrate to potassium in the diet exceeded 75:1, attacks were prone to occur. Increase of fat in the diet also predisposed toward attacks, but high protein diets had no such effect. Ingestion of excessive quantities of water or sodium chloride were without effect.

On the basis of these findings the authors recommend diets "medium high in both protein and fat and low in carbohydrate" and state that such diets have benefited both the patient studied and his siblings.

KAPLAN, Cincinnati.

NUTRITIONAL HYDROCEPHALUS IN INFANT RATS. L. R. RICHARDSON, Proc. Soc. Exper. Biol. & Med. **76**:142 (Jan.) 1951.

Four hydrocephalic rats, two males and two females, were reared to maturity, and litters were obtained by mating these animals to one another and to normal animals. The authors found that mature hydrocephalic rats were capable of producing young and that when the parents received a stock diet none of the offspring were hydrocephalic. It was found that in 7.48% of the young born to female rats receiving a purified diet low in folic acid hydrocephalus developed. The incidence was decreased to 0.15% upon addition of 200 γ of folic acid per 100 gm. of diet. The incidence was 2.02% when the diet contained 2 gm. of Merck's APF concentrate No. 3 per kilogram. When both folic acid and APF concentrates were added, the incidence was the same as when folic acid alone was added. The addition of 10 mg. of crude folic acid antagonist added to the basal diet which contained APF concentrate decreased the number of litters per female but did not increase the incidence of hydrocephalus.

HANSEN, Galveston, Texas.

VITAMIN B_{12}, A FACTOR IN PREVENTION OF HYDROCEPHALUS IN INFANT RATS. BOYD L. O'DELL, JAMES R. WHITLEY, and ALBERT G. HOGAN, Proc. Soc. Exper. Biol. & Med. **76**:349 (Feb.) 1951.

Female rats maintained from weaning to maturity on a ration containing soybean oil meal, Cerelose (glucose), lard, salt mixture, and all recognized vitamins except ascorbic acid, nicotinic acid, folic acid, inositol, and vitamin B_{12}, were compared with the findings on animals fed

stock diet. The experimental basal ration is low in vitamin B_{12} but does not show any other serious deficiency for growth in the rat. The incidence of hydrocephalus in the offspring of these animals, however, was 28%. The addition of vitamin B_{12} concentrate to the diet or the injection of crystalline vitamin B_{12} in the dams during early stages of gestation prevented the abnormality and increased their viability. Hydrocephalus was not prevented by folic acid alone, but it has not been determined whether vitamin B_{12} alone is effective or whether both folic acid and vitamin B_{12} are required to prevent this type of hydrocephalus in infant rats.

HANSEN, Galveston, Texas.

RADIOACTIVE IODINATED HUMAN SERUM ALBUMIN AS TRACER AGENT FOR DIANOSING AND LOCALIZING INTRACRANIAL LESIONS. SHELLEY N. CHOU, J. BRADLEY AUST, GEORGE E. MOORE, and WILLIAM T. PEYTON, Proc. Soc. Exper. Biol. & Med. **77**:193 (June) 1951.

Diiodofluorescein has given most satisfactory results of the various isotopes used to diagnose and localize intracranial tumors by the isotope-encephalometric technique. Radioactive iodinated human serum albumin (RIHSA) also has been used and appears to be as satisfactory, if not superior, to diiodofluorescein; 1 ml. contains 5 mg. of human serum albumin with a salt concentration of approximately 2 mg.; when injected intravenously into animals and human beings it remains in the blood stream for a long time. A focus of high uptake obtained by the initial counting after injection should persist through the second, third, and even the fourth day. This technique has been used in 10 cases up to date. The authors were able to localize tumors in five cases, subdural hematoma in one case, and rule out tumors in two cases in which subsequently air studies and angiograms were negative.

HANSEN, Galveston, Texas.

VENOUS THROMBOSIS IN ACUTE INFANTILE HEMIPLEGIC. R. G. MITCHELL, Arch. Dis. Childhood **27**:103 (Feb.) 1952.

Acute infantile hemiplegia is briefly described, and the findings in 10 cases are noted. Two of these cases showed features pointing to intracranial venous thrombosis as a cause.

The cause and effect of intracranial venous thrombosis are discussed, and illustrative cases are described.

It is concluded that venous thrombosis due to a minor upper respiratory infection may be the cause of many cases of this syndrome and that division of cases into those with and those without obvious infection is unnecessary.

Treatment with anticoagulants and antibiotics is advised in those cases in which the hemiplegia is due to venous thrombosis.

KELLY, Milwaukee.

EXAMINATION OF CEREBROSPINAL FLUID. C. K. PARIKH, Indian J. Child. Health **1**:350 (July) 1952.

The author merely outlines briefly the routine tests to be carried out on the cerebrospinal fluid in cases in which spinal puncture is performed.

MCGUIGAN, Evanston, Ill.

A FATAL CASE OF ENCEPHALITIS WITH UNILATERAL RHYTHMICAL MOVEMENTS ACCOMPANIED BY SYNCHRONOUS LOSS OF CONSCIOUSNESS. J. A. CHAVANY, G. GUIOT, and D. HAGENMULLER, Presse méd. **59**:1632 (Dec. 8) 1951.

A child of 9 years was stricken with encephalitis, apparently caused by a neurotropic virus. Within three months he was dead. The characteristic seizure consisted of slow sustained contractions of the left arm, coming on every 15 seconds and lasting 6 seconds. With each contraction, there was a loss of consciousness. The motions were much less marked while the child was lying down.

Detailed anatomical descriptions of the brain are given. The authors consider the condition to be a form of chorea, midway between Sydenham's chorea and von Economo's syndrome.

DAFFINEE, Melrose, Mass.

A Rare Case of Rachischisis with Multiple Malformations. Pierre-J. Rosselet, Schweiz. med. Wchnschr. **82**:12 (Jan. 5) 1952.

An infant born at term presented various malformations. It survived three days. There were hydrocephalus, a meningomyelocele and below this an area denuded of skin, covered with a mucosa with the histological features of rectal mucosa. Implanted in this mucosa was a small polyp which proved to be an aberrant adrenal gland. Abnormalities of the external sexual apparatus existed. The vertebral column was normal down to the fifth thoracic vertebra. From there on down to the first lumbar the vertebrae became enlarged, and from the first lumbar on the spinal column divided into two parts, each one formed by hemivertebrae; these rejoined each other terminally by means of a bridge formed by the first three left sacral hemivertebrae.

Amberg, Rochester, Minn.

Polyradiculoneuritis in an Infant, Complicated by the Unusual Finding of Periostitis. A. M. Cavallotti, Lattante **23**:556 (Sept.) 1952.

Quadriplegia and paralysis of the neck muscles gradually developed in a 2-month-old baby, a few days following the incision and healing of a staphylococcic abscess. He also had a periostitis of the femora, as seen by x-ray. A high temperature and globulin in the spinal fluid persisted for some time, in spite of penicillin and chloramphenicol treatment. However, the fever fell by lysis, and the periostitis cleared up after a course of Staphylococcus toxoid. The paralysis was still present one year later. A diagnosis of poliomyelitis was considered, but the final conclusion was that the child had polyradiculoneuritis with periostitis, possibly traceable to a Staphylococcus infection.

Higgins, Boston.

Cranial Defects. M. Crisalli, Lattante **23**:591 (Oct.) 1952.

A case report is presented of a newborn infant with spina bifida, meningocele, club feet, and defects in the bones of the cranium. The child died of a purulent meningitis at the age of 20 days, hydrocephalus having in the meantime developed.

Higgins, Boston.

Accidental Reactions Due to Penicillin Administered into the Subarachnoid Space. G. Maini, Lattante **23**:610 (Oct.) 1952.

Two babies, 6 and 8 months old, with Meningococcus meningitis had convulsions and hyperpyrexia following the administration of the sixth and seventh daily dose of 20,000 units of penicillin intrathecally. By this time the spinal fluid had become sterile. Penicillin given intramuscularly subsequently caused no reaction. The author cautions against the giving of penicillin intrathecally longer than is absolutely necessary.

Higgins, Boston.

Clinical Value of Encephalography in Infants. P. Durand and I. Scagliotti, Minerva pediat. **3**:678 (Dec. 1) 1951.

Pneumoencephalography in normal children under 2 years of age frequently reveals air pockets in the subarachnoid areas suggestive of cerebral atrophy or subarachnoid hydrocephalus. Repetition of the encephalogram after the child has passed his second birthday show that these airpockets no longer are to be found. Encephalograms of several infants with clinical signs of cerebral pathology showed similar airpockets, which disappeared after the child became 2 years old. A postmortem examination of one child who showed these airpockets revealed no pathological lesion which might explain the x-ray finding. Therefore, caution is necessary so that a diagnostic error is not made when one interprets an encephalogram of a young child.

Higgins, Boston.

Acute Ataxia of Leyden-Westphal. E. Cossandi and Carla Lombardi, Minerva pediat. **3**:693 (Dec. 1) 1951.

Fifteen days following a grippal infection, ataxia developed in a 2-year-old child, with loss of equilibrium both when standing and when walking. The child recovered from the ataxia

completely in about two weeks. A diagnosis of acute ataxia of the Leyden-Westphal type was made; the disease was probably caused by a virus infection in a susceptible child with a family history of neuropathic abnormalities.

HIGGINS, Boston.

TENDON AND CUTANEOUS REFLEXES IN NORMAL INFANTS. RICARDO OLEA G., Rev. chilena pediat. **22**:505 (Nov.) 1951.

The author believes that a better understanding of the normal characteristics of both superficial and deep reflexes in children under 3 years of age is needed. The long and patient investigations that are the basis for this knowledge do not lend themselves to being done by one person.

After a brief consideration of the basic concepts a report is made of the results obtained in 81 healthy infants (46 of whom were newborn).

Most of the tendon reflexes are present at birth but with different intensity, owing probably to the muscular tone proper to that age. The muscular tone continues to change with the maturation process, until a stage similar to that in the adult is reached, at a later age than the one studied in this article. Reflexes easily obtained in the newborn include the masseteric, bicipital, radial, midpubic, thigh adductor, patellar, Mendel-Bechterew, and Rossolimo. The abdominal muscle reactions are of a medium intensity. The midsternal, tricipital, and ankle reflexes are weak or difficult to obtain. Because of the physiological contracture of the pertinent muscles, the finger flexor and the cubital-pronator reflexes cannot be studied.

Some of these reflexes persist more or less the same during the age period studied in this article, but others increase or decrease. Those that remain more or less the same are the jaw reflexes, the biceps reflexes, midsternal reflexes, and knee jerks. Those that diminish are the the midpubic, abdominal, adductor and toe flexor reflexes. Those that increase are the radial, tricipital, and finger flexor reflexes, and the ankle jerks. The cubital-pronator reflex stands by itself; while the response of the shoulder abductors diminishes, the response of adult type begins to appear in some older infants. Diffusion of the response, as well as an increase of the reflexogenous zone, is common.

In a special group the peroneal and facial signs are considered. The former are nearly always negative and the latter frequently positive, specially in the newborn.

The superficial reflexes behave as follows: The pharyngeal and corneal reflexes are always present and active. More frequent and active but not constant is the anal reflex. All these reflexes persist more or less with the same characteristics during the age period studied. The abdominal and cremasteric reflexes begin in the newborn period as weak and inconstant, but with age become progressively more constant and active. The plantar reflex is practically always present, but the response is varied. Both in the newborn and in the older infant its response is more dorsiflexion than plantar flexion. The author believes that in those cases in which there is an extension response (dorsiflexion) this response is not similar to a positive Babinski sign in an adult with pyramidal tract lesions; thus there is no physiologic Babinski sign. Gordan's, Schaefer's, and Oppenheim's signs, which are said to be similar to the plantar reflex, are quite different from the true plantar reflex in their behavior. In the newborn they are all positive, while in the older infant they are nearly always absent or doubtful. The fact is stressed that in infants of all ages extension, to be expected if the signs were similar to the plantar reflex, is absent.

The author compares his results with those obtained by other authors and concludes that a systematic study of these reflexes in a large number of children with neurological lesions should be undertaken to determine the exact symptomatological value of the data obtained in his present study.

FROM THE AUTHOR'S SUMMARY.

SIGNIFICANCE OF MUSCLE ACTION CURRENT DETERMINATION IN INFANTILE PARALYSIS. GUNTER IHLENFELDT, Deutsche med. Wchnschr. **77**:206 (Feb. 15) 1952.

The Bayer Musculometer allows a rough estimation of muscle activity by means of a simple apparatus using the lighting up of a neon gas bulb on muscular activity. In the absence of any muscular function the bulb remains dark. If the bulb flickers on efforts at active motion, it is certain that motor unities capable of function are present. Insistent training can often accomplish much. In cases of poliomyelitis important prognostic data may be obtained.

AMBERG, Rochester, Minn.

Psychology and Psychiatry

SIBLING RELATIONSHIPS WITH SPECIAL REFERENCE TO THE PROBLEMS OF THE SECOND BORN. ALFRED E. FISCHER, J. Pediat. **40**:254 (Feb.) 1952.

The second-born child is likely to have problems, especially if he and his older sibling are males. These problems are emotional. They usually appear between the ages of 1 and 3 years. Parents of these children should be advised to divide their love and affection equally between the siblings, according to their needs. The younger child should more frequently enter into family affairs. Competition between the children should be avoided. Whenever possible, the children should be separated in play and nursery groups, and they should be encouraged to develop separate sets of playmates. The older child should be helped to understand the younger, with caution to permit the younger child to talk and play in his own fashion. And parents must be reminded that each child is different from others and that they themselves may be different now from what they were when their first child was born.

MACDONALD, Pittsburgh.

A METHOD OF ASSESSING MATERNAL EFFICIENCY FOR SOCIO-MEDICAL SURVEYS. E. HEAN THWAITES and IAN SUTHERLAND, Arch. Dis. Childhood **27**:65 (Feb.) 1952.

Maternal efficiency is defined as "the ability of a mother to cope with and care for her child." Emphasis is laid throughout on assessing the mother's actual achievement in relation to her child, rather than on her basic capacity or her intentions.

An index of maternal efficiency was first derived from five separate assessments of the mother and of the environment which she created for the child, these assessments being combined by the statistical technique of factor analysis.

After due consideration of this index it was decided to omit the assessment of maternal health and base a revised index upon four assessments only: (1) the state of the child, (2) the diet of the child, (3) the mother's attitude to the child, and (4) the state of the home. The standards of assessment are set out. At the same time it was decided that the technique for combining the assessments was unnecessarily complicated in the present instance and that it would be a practical advantage to replace it by a much simpler system.

KELLY, Milwaukee.

ENURESIS AS A PSYCHIATRIC PROBLEM. G. MORA, Minerva pediat. **4**:552 (July 15) 1952.

Sphincter control by the child is necessary for personal hygiene and social propriety. Many factors have been presented as the basis for the regression or continuance of the infantile status. When no organic defect is present, enuresis becomes a purely psychological problem. Intrafamilial, and especially maternal, reaction to the condition is frequently a basis for neurotic disturbances, not only in the child but even in other members of the group.

HIGGINS, Boston.

TREATMENT OF RETARDED CHILDREN WITH GLUTAMIC ACID AND PYRIDOXINE. S. CORBO, Minerva pediat. **4**:927 (Nov. 15) 1952.

A school group of 20 mentally retarded children, 5 to 9 years old, were studied over a period of six months. Eight children served as controls, and 12 children received 6 to 16 tablets daily, each containing 0.5 gm. of glutamic acid and 2 mg. pyridoxine. Psychometric tests were made before and after the six-month period. The average intelligence quotient of the control children increased from 0.72 to 0.80, and of the children receiving the tablets, from 0.73 to 0.85. Detailed descriptions of some of the individual tests are presented.

HIGGINS, Boston.

CONGENITAL MALFORMATIONS OF THE HEART IN MONGOLOID SUBJECTS. G. GRANATA, A. BENCINI and L. PARENZAN, Pediatria **60**:281 (May) 1952.

Twenty-four of the 56 mongoloid children examined gave evidence of congenital malformation of the heart. All mongoloid children, even when there is no established anatomical defect, seem to have some functional weakness of the heart and the circulation of the blood.

HIGGINS, Boston.

Diseases of Genitourinary Tract

SERUM COMPLEMENT IN ACUTE GLOMERULONEPHRITIS AND THE NEPHROTIC SYNDROME. KURT LANGE, LAWRENCE SLOBODY, FRANK CRAIG, GLORIA OGUR, JACOB OBERMAN and FRED LO CASTO, Pediatrics **8**:814 (Dec.) 1951.

The hypothesis that glomerulonephritis is a complement-binding antigen-antibody reaction was tested by determining complement titers. In a control series of 52 children, complement was found to range between 0.84 and 3.0 units, with an average of 1.68 units. In five children with acute glomerulonephritis, the complement titers were markedly diminished, from 0.08 to 1.0 unit with an average of 0.43 unit; the value of 1.0 was only found once, in a child who had a spontaneous remission. The complement titers in 9 cases of "nephrosis" or the nephrotic stage in children were also found to be low, ranging from 0.09 to 1.0 units, with an average of 0.57 units. No differences in complement titers were observed between the nephrotic stage of glomerulonephritis and the so-called pure lipid nephrosis. Complement levels return to normal approximately one week after the subsidence of clinical and laboratory signs of activity. Diuresis, whether spontaneous or induced by ACTH therapy, is accompanied by a return of complement to normal.

FROM THE AUTHORS' SUMMARY.

HYDROCOLPOS IN INFANCY AND CHILDHOOD. LEON ANTELL, Pediatrics **10**:306 (Sept.) 1952.

A case of hydrocolpos in an infant 6 days of age is reported, with a review of the literature. A new feature, consisting of areas of ecchymosis of the skin, is added. The source and nature of the fluid, the pathology, method of diagnosis, and prognosis and treatment of the condition are discussed.

FROM THE AUTHOR'S SUMMARY.

NEPHROTIC SYNDROME IN CHILDHOOD. J. G. WILSON, Clin. Rep. Adelaide Child. Hosp. **2**:112 (Nov.) 1951.

Thirteen cases of nephrosis were reviewed. Twelve patients recovered completely. No conclusions could be reached regarding effective treatment, and no addition to current knowledge was found in the 13 cases.

McGUIGAN, Evanston.

ENURESIS AND TOILET TRAINING. JOHN BOSTOCK and MARJORIE G. SHACKLETON, M. J. Australia **2**:110 (July 28) 1951.

The authors explored the relation of breast feeding and toilet training to later enuresis. No significant association with breast feeding was found. Rigid toilet training is strongly associated with later enuresis. There is a definite association between the type of training and acceptance of the child. Rigidly trained children are apt to be found to be unwanted.

McGUIGAN, Evanston, Ill.

SARCOMA OF THE BLADDER IN AN INFANT. A. PUGLIONISI and L. LEONARDI, Lattante **23**:577 (Oct.) 1952.

A 6-month-old boy began to have a fever and pyuria which did not respond to treatment. When he was admitted to a hospital two months later, a meaty mass was observed protruding from the penis; this mass could be withdrawn a distance of 4 cm. from the urethra. Suprapubic operation revealed a large egg-shaped tumor, which was diagnosed histologically as spindle-cell sarcoma. The child died two months later. The authors recommend total cystectomy and continued x-ray therapy for malignant growths of the bladder in children.

HIGGINS, Boston.

NEPHROTIC SYNDROME IN CHILDREN. ROANO ROSSI, Riv. clin. pediat. **50**:289 (May) 1952.

From his examination of the literature on the subject and from his observation of 17 cases of nephrosis syndrome, the author concludes that pure lipid nephrosis occurs very rarely. After

having delineated the limits of nephrotic syndrome—which is, after all, simply a particular manifestation of subacute glomerulonephritis—the author revises the various forms of therapy in use today with special reference to blood transfusion therapy.

From the Author's Summary.

Uropathy in Infancy: Presentation of Various Clinical Cases. J. L. de la Mano Marco, Acta pediat. espan. **118**:781 (Oct.) 1952.

Four case histories are presented representing exstrophy of the bladder in a 3-month-old girl, calculus of the bladder in a boy 3 years of age, calculus of the kidney in a 7-year-old boy, and tuberculosis of the kidney in a 6-year-old girl. Reproductions of x-rays are included in each case.

Diagnosis, treatment, and details of the surgical intervention are given. Intravenous urography has made diagnosis much simpler in the case of children. The various instruments of proper caliber and the operative procedures of de la Pena, Meredith Campbell, and others, have rendered intervention for congenital anomalies less dangerous. The antibiotics have lowered the chances of death by infection.

In all cases the children survived the operation successfully. In the first case uretero sigmoidotomy and removal of the bladder, which lacked an anterior wall, were done. Plastic ureteral catheters were expelled after six days. Such cases are rare and the patients frequently die within a few days, but this child had an uneventful recovery.

The child in the fourth case had the left kidney removed because of a tuberculous infection and also recovered. Among the therapeutic aids used were large amounts of vitamin A, streptomycin, and x-ray therapy to the area of incision, which healed slowly.

The author discusses the incidence of these urinary lesions in regions where diet is poor in vitamin A, as in China and India. Calculi in these countries are very common, whereas their incidence in the United States and certain other countries is very low.

Allen, San Francisco.

Disturbances of the Regulatory Mechanism of Circulation and Myocardial Damage in Acute Diffuse Glomerulonephritis in Childhood. H. Pluckthun and F. Hosemann, Ztschr. Kinderh. **69**:84 (No. 1) 1951.

The authors found cardiac involvement in 26 of 130 patients with acute glomerulonephritis. They feel that primary myocardial damage due to increased capillary permeability and perivascular edema are the causative factors involved.

Glaser, Jerusalem.

New Findings on the Diagnosis and Clinical Aspects of Chronic Aminoaciduria with Storage of Cystine. Helmut Weyers, Acta Pediat. **41**:334 (July) 1952.

Latest observations of a typical chronic amino-aciduria with storage of cystine in the 1-year-old, fair-haired daughter of parents related to each other have led to new diagnostic and clinical findings concerning this rare disease. Besides the older method of proving the presence of cystine crystals in smear preparations from the sternal marrow, the technically simple preparation of crystalline cystine in the conjunctiva of the eye can be used to confirm a suspected diagnosis. In crystallographic differentiation two kinds of crystal formations may be distinguished, differing from each other by a characteristic refraction. Chromatographic estimations of amino acids are compared with microbiologically ascertained concentrations of amino acids in the urine and with the less known serum estimations. Although there are varying concentrations of other amino acids there is no increase of cystine in either serum or urine. The storage of the almost insoluble cystine in the reticuloendothelial system is confirmed by electrophoretic estimation, proving that a transposition of serum-proteins has taken place. As far as the glucose lability of this disease is concerned, new transpositions of the potassium balance connected with the metabolism of glucose are discussed. It is doubtful whether the excretion of reducing substances which could be explained by the accumulation of acid compounds in the blood of

children suffering from cystinosis is of the glucose type, since there is a discrepancy between findings on polarimetry and on decisive fermentation tests. Special emphasis is placed on the close affinity between amino-aciduria of the cystinosis type and celiac disease. This is based on the similar pathogenesis and clinical picture.

FROM THE AUTHOR'S SUMMARY.

Diseases of Ductless Glands; Endocrinology

PRODUCTION OF EXPERIMENTAL CRETINISM IN DOGS BY ADMINISTRATION OF RADIOACTIVE IODINE. CURTIS A. SMITH, HARRY A. OBERHELMAN JR., EDWARD H. STORER, EDWARD R. WOODWARD, and LESTER R. DRAGSTEDT, A. M. A. Arch. Surg. **63**:807 (Dec.) 1951.

Experimental cretinism may be produced in dogs by administration of I^{131} to the pregnant bitch prior to whelping or to the newborn dog during the first few months of life. Cretinism in dogs is characterized by impairment of growth and skeletal development, obesity, lethargy, coarse dry skin and hair, and, occasionally, protuberant tongue. Cretin dogs have hyperlipemia and hypercholesteremia, and conversion of free to protein-bound iodine is depressed. Associated endocrine changes are the immaturity of the gonads and increase in size of the pituitary.

FROM THE AUTHORS' SUMMARY.

EOSINOPHILIA IN THE THYMUS: A SIGN OF ADRENAL CORTICAL INSUFFICIENCY IN INTRAPARTUM AND NEONATAL DEATH? ROBERT W. HILLMAN, Brooklyn Hosp. J. **9**:158, 1951.

During the year 1946 autopsies were performed on 53 infants who were stillborn or who died in the neonatal period. Sections of the thymus were taken in 21 cases; of these, 12 sections were judged to contain an excess of eosinophiles in varying degrees from 1 + to 4 +. In cases in which the most pronounced eosinophilia was present intracranial hemorrhage seemed to occur more frequently. The author considers the phenomenon to be consistent with and even suggestive of adrenal cortex hypofunction. It may represent a fatal deficiency of hormones in the fetus in cases of neonatal or intrauterine death.

The author believes that correction of adrenal insufficiency may be the best means of reducing mortality and morbidity in this age group.

McGUIGAN, Evanston, Ill.

EFFECT OF STEROIDS AND LACTONE DERIVATIVES ON HYPHOPHYSEAL GONADOTROPIN CONTENT. GLENN E. MORTIMORE, C. ALVIN PAULSEN, and CARL G. HELLER, Endocrinology **48**:143 (Feb.) 1951.

The search for exogenously administered agents which will control pituitary gonadatropic function has revealed that inhibition may be produced by estrogens, androgens, and progesterone when large doses are given. Mild stimulation may result from the administration of small amounts of some of these steroids. Clinical application of these agents when pituitary suppression or stimulation is desired is limited by their marked sex-hormone effects. For example, it would be ideal to cut off pituitary stimulation to the ovary in the presence of endometriosis or premature puberty. However, (1) estrogens cannot be used because amounts large enough to suppress pituitary gonadotropins aggravate these conditions; (2) androgens are limited in usefulness by their virilizing effects, and (3) the dose of progesterone is too large to be practicable. No potent hypophyseal stimulator is known. Therefore, a number of substances unlikely to exhibit marked estrogenic or androgenic activity were screened to determine their ability to depress or stimulate hypophyseal gonadotropic output, at the same time assessing their estrogenic activity. Of the 14 steroids tested, not one prevented the postcastration rise in gonadotropins.

JACOBSEN, Buffalo.

EFFECT OF ADRENAL STIMULATION WITH ADRENOCORTICOTROPHIC HORMONE ON THE EXCRETION OF DEHYDROISOANDROSTERONE IN MAN. RICHARD L. LANDAU, KATHRYN KNOWLTON, KATHLEEN LUGIBIHL, and ALLAN T. KENYON, Endocrinology **48**:489 (May) 1951.

It was found that the excretion of urinary dehydroisoandrosterone and related ketonic and nonketonic chromogens paralleled the rise in 17-ketosteroids when the normal adult adrenal

cortex was stimulated with purified adrenocorticotropic hormone. A quanitative comparison of the increased excretion of ketonic Pettenkofer chromogens (dehydroisoandrosterone) and 17-ketosteroids following the administration of dehydroisoandrosterone acetate with the rise in these urinary steroids induced by adrenal stimulation in the same subject supports the suggestion that dehydroisoandrosterone and its metabolic and hydrolytic derivatives account for the major portion of the 17-ketosteroids of adrenal origin. Accordingly, shifts in the daily excretion of the Pettenkofer chromogens (dehydroisoandrosterone and related compounds) in a given subject may accurately reflect alterations in adrenal activity.

JACOBSEN, Buffalo.

DETERMINATION OF URINARY DEHYDROISOANDROSTERONE IN NORMAL MEN AND WOMEN AND IN ADRENAL DISEASE. RICHARD L. LANDAU, KATHRYN KNOWLTON, KATHLEEN LUGIBIHL and PAUL L. MUNSON, Endocrinology **48**:506 (May) 1951.

It was estimated that from 1.4 to 12.4 mg. per day of dehydroisoandrosterone was excreted by a series of normal men and women. The ketonic Pettenkofer chromogens thus comprised from 10 to 63% of the 17-ketosteroids in these persons. In general, the excretion of Pettenkofer chromogens fits well with other evidences of adrenal activity.

JACOBSEN, Buffalo.

EFFECT OF ADRENO-CORTICAL EXTRACTS ON BONE MARROW EOSINOPHILES OF MICE. M. L. DURGIN and ROLAND K. MEYER, Endocrinology **48**:518 (May) 1951.

Adrenalectomized male mice after injections with lipoadrenal cortex showed in the bone marrow a marked increase in younger stages of eosinophilic cells. The increase in activity of the bone marrow seems sufficient to explain the increase in eosinophiles in peripheral blood during recovery from the response to adrenal cortex extract.

JACOBSEN, Buffalo.

EFFECTS OF ANDRENOCORTICOTROPHIC HORMONE ON BODY GROWTH, VISCERAL PROPORTIONS, AND WHITE BLOOD CELL COUNTS OF NORMAL AND HYPOPHYSECTOMIZED MALE RATS. C. WILLET ASLING, W. O. REINHARDT and CHOH HAO LI, Endocrinology **48**:534 (May) 1951.

Growth was retarded in a group of intact rats by a dose of 2.0 mg. of corticotropin (ACTH) daily, and brought to a stop by 3.0 mg. daily. The hypophysectomized rats were even more stunted than their controls with a dose of 2.0 mg. daily. Regeneration of hair in shaved areas did not occur in the hypophysectomized treated rats. As judged by roentgenograms, skeletal maturation was neither accelerated nor retarded by the same treatment. Confirmation is presented for the lymphocytopenic and eosinopenic action of corticotropin as seen in chronic administration of corticotropin.

JACOBSEN, Buffalo.

EFFECT OF ACUTE AND CHRONIC DESOXYCORTICOSTERONE ACETATE PRETREATMENT ON THE PITUITARY-ADRENAL RESPONSE TO STRESS. C. E. HALL, J. C. FINERTY, O. HALL, and M. HESS, Endocrinology **48**:591 (May) 1951.

The finding that the depletion of adrenal ascorbic acid which normally ensues in response to various types of stress may be prevented or inhibited by pretreatment of the animal with cortical extracts and certain of the cortical steroids, in proportion to the dosage employed, has led to the concept of a "peripheral-humoral" control of pituitary-adrenal activity. This postulates that corticotropin (ACTH) is discharged by the hypophysis in response to the declining level of circulating cortical hormones, which follows stress as a result of increased tissue utilization or destruction of these steroids. In the experiments reported it was found that desoxycorticosterone when given either acutely or chronically fails to impair the ability of the pituitary-adrenal axis to respond to stress with the usual drop in adrenal ascorbic acid values.

JACOBSEN, Buffalo.

Growth-Inhibiting Effect of Cortisone Acetate on the Chick Embryo. David A. Karnofsky, Lois P. Ridgway, and Priscilla A. Patterson, Endocrinology **48**:596 (May) 1951.

It has been noted that 11-dehydro-17-hydroxycorticosterone-21-acetate (cortisone acetate) causes a striking inhibition of embryonic growth and development. This report presents further studies on the growth-inhibiting effects of cortisone when injected into the yolk sac or onto the chorioallantoic membrane of the chick embryo at various stages of development.

The mechanisms of action of cortisone on the chick embryo are unquestionably complicated, and two types of effects seem probable: (1) a local inhibitory effect, as evidenced by the action of cortisone applied locally to the chorioallantoic membrane of the four-day embryo in temporarily inhibiting the growth of the membrane and the embryo, and (2) the generalized and prolonged inhibition of embryonic development which appears to be initiated at the 8th and 10th days of development. In searching for an explanation for the onset of the "cortisone effect" of the embryo on the eighth day, it seems unlikely that cortisone acts primarily by inhibiting or modifying the function of the endocrine glands or that at this period the embryonic tissues abruptly become susceptible to the action of cortisone. More promising suggestions are that cortisone may either specifically inhibit the formation of a new function or tissue, essential for growth, normally appearing at the 8th to 10th day, or interfere with the utilization of a new substrate or substrates which are provided for cell growth as a result of the shift in embryonic metabolism.

Jacobsen, Buffalo.

Inhibition of DCA Induced Hypertension and Hyalinosis by a Drug Acting on Membrane Permeability. Roger Guillemin and Claude Fortier, Endocrinology **48**:617 (May) 1951.

The sodium content of the diet has been conclusively shown to be an essential factor in the pathogenesis of desoxycorticosterone acetate-induced hypertension and associated morphological alterations, since such changes are prevented on a salt-free diet. Furthermore, high doses of MaCl are conducive in some species to hypertension and hyalinosis even in the complete absence of mineralocorticoids. The Na ion would thus seem to be the main responsible agent, salt-retaining steroids merely facilitating its action. This facilitating role of desoxycorticosterone acetate and related steroids could be ascribed to altered membrane permeability, leading to an increased content of intracellular sodium at the level of the proximal convoluted tubules. A so-called antihistaminic compound promethazine ([1-dimethyl-amino-2'-methyl-1'-ethyl-N-] dibenzo-*p*-thiazinehydrochloride), also known as 3277 R. P. or Phenergan, has been shown to have opposite effects on membrane permeability. It therefore appeared of interest to determine whether its administration could prevent the development of hypertension and associated morphological lesions in animals treated with desoxycorticosterone acetate. It was found that the changes were satisfactorily prevented.

Jacobsen, Buffalo.

A Quantitative Method for the Bioassay of the Effect of Adrenal Cortical Steroids on Mineral Metabolism. S. A. Simpson and J. F. Tait, Endocrinology **50**:150 (Feb.) 1952.

A method is described for the quantitative measurement of the ability of substances to restore the normal electrolyte balance in adrenalectomized rats. The application of such a method in the isolation of active principles in cortical extracts and in the testing of synthetic compounds is suggested.

Jacobsen, Buffalo.

Effects of Purified Gonadotrophins on the Cholesterol Content of the Testes of Immature Rats. Jay Tepperman and Helen M. Tepperman, Endocrinology **50**:162 (Feb.) 1952.

A purified pituitary follicle-stimulating hormone preparation was without effect on the cholesterol ester content of the testes of intact weanling rats. Injection with several purified

pituitary luteinizing hormone preparations markedly increased the cholesterol ester content of the testes of comparable animals. It is concluded that only those materials which contain luteinizing hormone of pituitary origin caused an increase in testis cholesterol ester.

JACOBSEN, Buffalo.

ENHANCEMENT OF ADRENOCORTICOTROPIC HORMONE ACTIVITY BY ALUM IN NORMAL 21-DAY-OLD MALE RATS. T. HAYASHIDA and CHOH HAO LI, Endocrinology **50**:187 (Feb.) 1952.

The alum-precipitated preparation of highly purified corticotropin (ACTH) protein has been investigated with respect to its ability to cause adrenal hypertrophy and thymus involution in 21- to 23-day-old normal male rats. It was found that the adrenocorticotropic activity of the hormone was definitely enhanced by alum treatment, presumably through the effect of delayed absorption.

JACOBSEN, Buffalo.

A GLYCOSTATIC EFFECT OF TESTOSTERONE ON THE PERINEAL MUSCLES OF THE RAT. SAMUEL L. LEONARD, Endocrinology **50**:199 (Feb.) 1952.

It is known that the striated muscles of the perineal complex of the male rate decrease in size after castration and increase in size under the influence of male hormone.

In this paper it is shown that castration is accompanied by a significant decrease in glycogen in these muscles and that injections of testosterone propionate results in a marked increase. It is also demonstrated that the effect of testosterone on the glycogen levels of these muscles is independent of the pituitary.

JACOBSEN, Buffalo.

WATER EXCRETION IN HYPOPHYSECTOMIZED RATS BEFORE AND AFTER ADRENALECTOMY. FREDERICK G. HOFMANN, Endocrinology **50**:206 (Feb.) 1952.

The hypophysectomized rat excretes a test-load of water more slowly than does an intact animal. The rate of excretion is further and markedly reduced after adrenalectomy, indicating that the adrenal cortex does possess some degree of function even in the absence of the anterior pituitary.

JACOBSEN, Buffalo.

INACTIVATION OF ADRENOCORTICOTROPIC HORMONE IN VITRO BY TISSUES. IRVING I. GESCHWIND and CHOH HAO LI, Endocrinology **50**:226 (Feb.) 1952.

The fact that frequent injections of corticotropin (ACTH) are necessary in order to achieve a maximum physiological or clinical effect, along with the rapid disappearance of adrenocorticotropic activity from plasma and absence of activity in the urine, indicates that the hormone is rapidly destroyed or utilized in the organism. In order to determine the contributions of various tissues to the loss of corticotropin activity, a study was made of the inactivation of the adrenocorticotropic hormone in vitro by organ slices and homogenates.

It was found that, with the probable exception of plasma, all the tissues studied were capable of causing inactivation of corticotropin. Several possible reasons for this are discussed.

The authors hypothesized that the ability of tissue to inactivate corticotropin, even after heating, can be explained by the binding or absorption of the hormone to the particular tissue.

JACOBSEN, Buffalo.

SOME EFFECTS OF CORTISONE ON VITAMIN C-DEFICIENT GUINEA PIGS. E. H. HERRICK, E. RUTH MEAD, BARBARA W. EGERTON, and J. S. HUGHES, Endocrinology **50**:259 (Feb.) 1952.

In guinea pigs from which vitamin C was withheld the usual conditions of scurvy with painful joints and degenerating bones and testes developed; other guinea pigs that received no vitamin C but were given 5 mg. of cortisone acetate daily lived from 17 to 45 days on the scorbutic diet and had no painful joints but showed adrenal glands with atrophied cells, shrunken nuclei, few cytoplasmic granules, and decreased lipoidal vacuolation.

JACOBSEN, Buffalo.

Identification of Pituitary Interstitial Cell Stimulating Hormone in Human Urine. Janet W. McArthur, Endocrinology **50**:304 (March) 1952.

A method for the preparation and identification of pituitary interstitial cell-stimulating hormone in human urine is described.

Jacobsen, Buffalo.

Histochemical Demonstration of Ketosteroids in Adrenal Cortical Tumors With or Without an Associated Cushing's Syndrome. Arnold M. Seligman and Rivka Ashbel, Endocrinology **50**:338 (March) 1952.

Adrenal cortex tumors associated with Cushing's syndrome which elaborate large amounts of the corticosteroids 17-hydroxy-11 dehydrocorticosterone and 17-hydroxycorticosterone (compounds E and F) may show no ketonic carbonyl groups (ketosteroids) when the tissue is fixed in formalin for four years or longer. On the other hand, adrenal cortex tumors associated with precocious development and virulism give positive reactions for ketonic carbonyl groups (ketosteroid) even after fixation in formalin for as long as 11 years.

Jacobsen, Buffalo.

Clinical Evaluation of Corticotropin Therapy in Children. Marshall B. Kreidberg, Alfred P. Cleroux, and Isadore N. Rosenberg, J. Pediat. **39**:397 (Oct.) 1951.

Corticotropin (ACTH) was helpful in bronchial asthma, nonthrombocytopenic purpura, dermatitis venenata, rheumatic fever, Sydenham's chorea, rheumatoid arthritis, and nephrosis. Subacute lymphatic leukemia was temporarily modified. Not affected by corticotropin were glomerulonephritis, dermatitis exfoliativa (Wilson's disease), polymyositis, and thrombocytopenic purpura.

Macdonald, Pittsburgh.

Mixed Adrenal Disease of Infancy. Alfred G. Knudson Jr., J. Pediat. **39**:408 (Oca.) 1951.

Knudson reports 11 cases of mixed adrenal disease in infancy. He presents evidence for the hereditary nature of the disease; he believes that it is inherited by recessive genes. The fundamental defect, he suggests, is in adrenal enzymes with resultant disturbed steroid metabolism

Macdonald, Pittsburgh.

Adrenal Cortical Hyperfunction in Childhood: Report of a Case with Adrenocortical Hyperplasia and Testicular Adrenal Rests. Elizabeth Kirk Rose, H. T. Enterline, J. E. Rhoads, and Edward Rose, Pediatrics **9**:475 (April) 1952.

A case of adrenocortical hyperfunction occurring in a boy aged 3⅔ years is reported. The clinical features included bulimia, obesity, hypertension, hirsutism, personality change, osteoporosis, and intermittent neurologic manifestations. The urinary excretion of 17-ketosteroids and corticoids was increased. There was no genital dystrophy or impairment of glucose tolerance. Marked temporary improvement followed partial bilateral resection of the enlarged adrenals. The patient died 10 months after the operation, after exposure to epidemic parotitis, as a result of massive hemorrhage from ulceration of the esophagus. Necropsy showed bilateral nodular adrenocortical hyperplasia limited to the fascicular and reticular zones. Bilateral rests of adrenocortical cells were found in the rete testis. Neither Crooke's changes nor adenomata were found in the basophile cells of the anterior pituitary.

From the Authors' Summary.

Follow-Up Study of Juvenile Diabetics. R. B. Kerr, G. P. Brown, and N. Kalant, Canad. M. A. J. **66**:97 (Feb.) 1952.

The authors studied a group of eighty-one diabetic patients in whom diabetes mellitus developed in childhood or in early adolescence and who had had the disease for periods up to 28 years.

In 40% of the group reasonably good control had been maintained. Of the 81 patients, 49 had demonstrable complications; 41 had retinopathy, 7 had cardiovascular disease, 11 had renal disease, 13 had vascular calcifications, 8 had neuropathy, and 8 had skin lesions. As would be expected, the incidence of complications was an index of the degree of control of the diabetes.

There were 23 pregnancies in nine diabetic women, in four of whom the disease was well controlled and in five poorly controlled. In 10 pregnancies among the well-controlled group there were no abortions, there was one fetal or neonatal death and there were nine living children. In the other group there were 13 pregnancies with nine abortions, two fetal or neonatal deaths and two living children.

Four of the 81 patient died during the study; in all the disease had been poorly controlled. Average duration of diabetes in the four was 22 years.

McGUIGAN, Evanston, Ill.

CRETINISM. MARGARET MULLINGER, JOHN MUNN, and A. D. CHUTE, Canad. M. A. J. **66**:560 (June) 1952.

Findings in 22 cases of cretinism are very briefly discussed and treatment suggested.

McGUIGAN, Evanston, Ill.

THE HAND-SCHÜLLER-CHRISTIAN SYNDROME. G. LAGOMARSINO and M. MERLINI, Minerva pediat. **3**:646 (Nov. 15) 1951.

The case of a 4-year-old girl with Hand-Schuller-Christian syndrome was followed over a period of four years in the Pediatric Clinic at Genoa, and many studies as to her growth, dentition, vegetative nervous systems, and body chemistry, as well as her clinical course, are recorded.

HIGGINS, Boston.

X-RAY PICTURE OF BONE CHANGES IN A CASE OF THYROID DWARFISM. G. ROTTINI, Minerva Pediat. **3**:697 (Dec. 1) 1951.

A cretin with a chronological age of 14 years and a mental age of 8 months was found by x-ray examination to have many marked skeletal abnormalities; several of these abnormalities have not been reported in the medical literature.

HIGGINS, Boston.

PATHOGENESIS OF DIABETES MELLITUS. MEJ. C. VAN BEEK, Maandschr. kindergeneesk. **19**:85 (May) 1951.

A historical review of the pathogenesis of diabetes mellitus is given. The islets of Langerhans do not form a unit, either in a morphological or in a functional respect. Three types of cells are observed in the human islands. The B cells greatly outnumber the A cells, and only a very few D cells are present. The B cells are probably concerned with insulin production. Quantitative as well as qualitative changes may be observed in the islets in diabetes mellitus. In young patients atrophy is the most notable lesion. After middle life atrophy and especially local amyloidosis of the islets are often seen. There exists a parallelism between amyloid deposition and arteriosclerosis, which may be causative. It is suggested that this amyloid deposition may be due to insulin which has insufficiently passed the capillary wall. This suggestion finds support in observations made on insulinomas, in which a positive amyloid reaction was sometimes obtained. Hydropic degeneration is not a cause of diabetes but is always secondary; it is a sign of exhaustion after initial stimulation by hyperglycemia. That this change is rarely seen nowadays, in contrast with former times, may be the consequence of insulin treatment, which protects the B cells against severe hyperglycemia. The presence of hydropic degeneration sometimes observed in insulinomas may be the result of hyperglycemia which existed before and during operation owing to intravenous dextrose administration.

It has recently been shown that the B-A ratio in diabetes is very low or B cells are absent altogether, even in cases in which the islets look normal with routine stains. In diabetes mellitus, examination of the pancreas is therefore incomplete unless special staining techniques

are carried out and an island cytogram made. Since experiments have been described from America and Germany which have provided very strong presumptive evidence that a hyperglycemic and glycogenolytic hormone is secreted by the A cells, the horizon is widened. Indications for future investigations are given.

FROM THE AUTHOR'S SUMMARY.

Diseases of Muscles, Bones, Joints, and Lymph Nodes

MYOSITIS OSSIFICANS PROGRESSIVA. HARRIS D. RILEY JR. and AMOS CHRISTIE, Pediatrics **8**:753 (Dec.) 1951.

Four new cases of myositis ossificans progressiva are presented and the literature on the subject is reviewed. At the onset in typical cases palpable masses will usually develop over the fascial planes of the head and neck. Biopsy of these lesions suggests tumor tissue, but this subsequently goes on to calcification and ossification. This point illustrates that the disease title —myositis ossificans—is really a misnomer, since there is no calcification of muscles themselves but rather a calcification of the connective tissue between the muscles.

When the disease progresses as illustrated by the cases reported the patients frequently present themselves with gait or posture disturbance or with other orthopedic complaints. Microdactylism and hallux valgus deformity of the great toe are two of the outstanding findings.

The cases reported illustrate the various stages of the disease under discussion. From inflammation, swelling, and induration of the muscles and connective tissues to tumefaction, the disease finally progresses to calcification in two to eight months. Finally, the calcification progresses to ossification and severe crippling. Serial illustrations and roentgenograms on several of these cases illustrate these progressive changes.

All biochemical determinations have been normal, but marked subjective improvement following cortisone therapy in one early case suggests the possible relationship of this disease process to disturbed steroid metabolism.

FROM THE AUTHORS' SUMMARY.

GROWTH OF THE FIBULA OF PREMATURE INFANTS AS ESTIMATED IN X-RAY FILMS: A METHOD FOR ASSESSING FACTORS PROMOTING OR INHIBITING GROWTH. RICHARD DAY and WILLIAM A. SILVERMAN, Etude Neo-Natales **1**:111 (Dec.) 1952.

An x-ray technique is described which measures the length of the fibula of premature infants. The coefficient of variation is 0.2%. This degree of accuracy makes possible a rapid evaluation of growth-promoting or growth-retarding factors.

FROM THE AUTHORS' SUMMARY.

A CASE OF HURLER-PFAUNDLER'S DISEASE. A. M. CAVALOTTI, Pediatria **60**:357, 1952.

The Hurler-Pfaundler syndrome differs from other types of dysostosis by reason of the storage of a lipid substance, not yet identified, in the cartilage as well as in several other organs. The case described is that of a mentally deficient 4-year-old boy, with a large liver and spleen and deformities. One interesting finding was an eosinophilia in both the peripheral blood and the bone marrow smear, although there were no intestinal parasites found. The mother and a sister of the patient also had eosinophilia, although both were otherwise normal.

HIGGINS, Boston.

CRANIOLACUNIA AND CRANIOFENESTRIA IN THE NEWBORN INFANT. V. KOTTOVA I. VONDRAKOVA, Pediat. listy **7**:337 (Dec.) 1952.

Craniolacunia and craniofenestria are congenital anomalies manifesting themselves on palpation as focal softenings to complete absence of bone in certain parts of the skull, while the ossification of the other skull bones and the rest of the skeleton remain intact. Roentgenographic examinations of the skull in anteroposterior and lateral views reveal shadow defects of varying shapes and sizes, hemmed by thickened bone. Other findings, including the levels and ratio of calcium phosphorus, do not deviate from the normal. These anomalies may occur singly or multiply, alone or in association with other anomalies. The authors discuss from their personal experience 14 cases of craniolocunia and 3 of craniofenestria. They were found in 0.67% of a total of 2,047 newborn infants examined.

FROM THE AUTHOR'S SUMMARY.

Diseases of Eye

Clinical Electroretinography in the Ophthalmological Examination of the Young Child. H. E. Henkes, Maandschr. kindergeneesk. **20**:300 (Oct.) 1952.

The author discusses some aspects of clinical electroretinography, especially in connection with the ophthalmological examination of the infant. From the Author's Summary.

Cutaneous Diseases; Allergy

Hapamine in the Treatment of Atopic Eczema. Aaron Nisenson, J. Pediat. **39**:472 (Oct.) 1951.

Hapamine (histamine-protein complex) was beneficial in 16 of 30 cases of intractable infantile eczema.

Macdonald, Pittsburgh.

Incontinentia Pigmenti: Report of Three Additional Cases. Adolph B. Loveman and Maurice T. Fliegelman, J. Pediat. **40**:442 (April) 1952.

Incontinentia pigmenti is a rare anomaly of skin pigment. It usually appears at birth or in infancy. Although it seems to be a congenital anomaly, it sometimes follows inflammatory processes of the skin. It is characterized by bizarre, irregularly shaped areas of slate or brown pigmentation on the trunk or extremities. The face is usually spared. The pigmented areas may be spidery or may resemble the colored veins of marble. Lesions occur soon after birth, usually in females. There is spontaneous disappearance after many years.

Macdonald, Pittsburgh.

Eczema in Children and Iodine Fixation in the Blood. D. V. Bori and G. Bellieni, Minerva pediat., **4**: 901 (Nov. 15) 1952.

The Hinsberg method was used to determine the blood-iodine fixation of 19 infants with eczema. It was found to be lower than normal in 14 infants, a finding which suggests that there is a tendency for the unsaturated fat of the blood of eczematous children to be low.

Higgins, Boston.

Leucolycytosin in the Blood During Serum and Histamine Shock. Prof. Franco Recchia, Luigi Ravazzoni, and Marcello Ghiron, Pediatria **59**:634, 1951.

In this work are reported the experiments performed on rabbits in order to show the eventual presence of leucocytolysines in the blood serum during serum and histamine shock.

From the experimental data concerning these two kinds of shock, the presence of leucocytolysines which in vitro cause a leucocytolysis (up to 42.1%) is evidenced. Leucocytolysines are destroyed by heat.

They gradually disappear in blood serum in the days following the shock; instead there appear leucocytolysine antibodies.

This work, besides pointing out the close analogy between leucopenia produced by histamine shock and that caused by serum shock, shows also a certain specificity of the leucocytolysines and evidence that the leucopenia must be regarded as a lysis of leucocytes caused by leucocytolysines formed during anaphylactic shock. From the Authors' Summary.

Pustular Psoriasis. Jorge Rosselot, Rev. chilena pediat. **21**:528 (Nov.) 1951.

The author stresses the clinical aspects of pustular psoriasis, a special form of psoriasis consisting of an association of recurrent pustular episodes, with special characteristics. He insists on the primary and fundamental characteristics that the pustular phenomena acquire in this variety of psoriasis that determine the nosological autonomy of this condition.

The rarity of the condition at any age is pointed out, more so in infancy, in which psoriasis itself is infrequent. He points out that the condition does not respond to treatments that sometimes effect recovery from simple forms of psoriasis.

The clinical history of a 7-year-old-child with clinical and histological lesions characteristic of pustular psoriasis is given in detail. There was complete recovery from this condition after prolonged administration of antibiotics (local and general treatment with penicillin and sulfathiazole) and massive doses of vitamin D_2. The patient was kept under observation for two years after recovery. There was no evidence of relapse.

FROM THE AUTHOR'S SUMMARY.

EPIDEMIC OF MONILIA ALBICANS INVOLVING THE SKIN SIMULATING LEINER'S DISEASE (DERMATITIS DESQUAMATIVA). J. B. MAYER, Monatsschr. Kinderh. **99**:88 (March) 1951.

The author observed an epidemic of extensive intertriginous lesions occurring in 40 infants; the lesions also involved the scalp and progressed to resemble Leiner's disease. The lesions always began in the region of the anus. Of 55 patients whose skin and stools were examined bacteriologically Monilia albicans was found in 30 in the feces and in 17 on the skin. The incubation period varied from two to four weeks; the disease was noted only in infants below 5 months of age. Clinically, differentiation between seborrheic dermatitis and Leiner's disease was impossible. Treatment with potassium permanganate baths and 5% boric acid powder was effective in all cases.

HARTENSTEIN, Milwaukee.

PROBABLE BACTERIAL-ALLERGIC ASTHMA IN CHILDREN AND ITS TREATMENT. E. WINGE FLENSBORG, GRETHE NEERBORG, and T. SAMSØE-JENSEN, Ugesk. læger **112**:387 (March 23) 1950.

In two-thirds of asthmatic children the attacks are preceded by catarrhal infection. A certain number of asthmatics do not react at all to routine allergen tests. Treatment with bacterial vaccines gives better results in these children than so-called specific hyposensitization in other asthmatic children. Sometimes, a very severe general reaction may occur after injection of bacterial vaccines. The authors are of the opinion that these facts indicate that the course of asthma in these children is a bacterial allergy, which, however, cannot be demonstrated by means of cutaneous tests. The asthmatic disease due to bacterial allergy is in all other respects identical with the ordinary asthma with manifestations such as hypersensitivity to cutaneous tests with alimentary and respiratory allergens.

WALLGREN, Stockholm, Sweden.

Teeth and Dentistry

SELENIUM AND DENTAL CARIES: AN INVESTIGATION AMONG SCHOOL CHILDREN OF OREGON. DEMETRIOS M. HADJIMARKOS, CLARA A. STORVICK, and LEMAR F. REMMERT, J. Pediat. **40**:451 (April) 1952.

The authors suggest that in some unknown manner the trace element selenium, found in some western soils, may contribute to dental caries.

MACDONALD, Pittsburgh.

FRACTURES OF THE DISTAL PART OF THE FOREARM IN CHILDREN. RAYMOND G. GIBERSON and JOHN C. IVINS, Minnesota Med. **35**:744 (Aug.) 1952.

The lower third of the forearm is perhaps the most frequent site of fracture in children. It has been stated that the younger the child the nearer the fracture is to the end of the bone, the greater will be the power of correction. Fractures in the middle third of the forearm need accurate alignment in all ages, since growth does not influence the angulation so greatly at this level.

The study was followed up in 15 children who had fractures of the distal third of the forearm and in whom there was residual deformity of the forearm at completion of treatment. These forearms eventually became practically normal in all respects. The authors stress that all possible efforts should be made to restore anatomic relations in the growing child, but repeated manipulation of these fractures are rarely necessary because growth will correct a remarkable amount of deformity.

STOESSER, Minneapolis.

Miscellaneous

STRAMONIUM POISONING. FRANK C. STILES, J. Pediat. **39**:354 (Sept.) 1951.

Datura stramonium grows wild in many parts of the U. S. This Milwaukee boy ate some, "the seeds of a thorn-apple," found in a vacant lot. He acted as if he were "intoxicated." Gastric lavage, a cathartic, a barbiturate, and intravenous dextrose resulted in complete recovery.

MACDONALD, Pittsburgh.

CASE OF ADRENOGENITAL SYNDROME WITH HYPERTENSION TREATED WITH CORTISONE. THOMAS H. SHEPARD and SAMUEL W. CLAUSEN, Pediatrics **8**:805 (Dec.) 1951.

A case of adrenogenital syndrome due to adrenocortical hyperplasia proved by biopsy and with the clinically unusual finding of hypertension has been treated with intramuscular injections of cortisone. Results of the treatment were reduction of growth, correction of the hypertension, and drop in urinary 17-ketosteroids. The syndrome may be related to excessive production of corticotropin. The hypertension in this case may be caused by hypertrophy of the zona glomerulosa of the adrenal cortex.

FROM THE AUTHORS' SUMMARY.

CHLOROMYCETIN PALMITATE: ITS USE IN PEDIATRICS. VINCENT DEPAUL LARKIN, Proc. Soc. Exper. Biol. & Med. **78**:191 (Oct.) 1951.

Esters of chloramphenicol (Chloromycetin) with higher fatty acids were prepared in order to attempt to produce a palatable form of the drug. Animal studies indicated that chloramphenciol with palmitic acid was less toxic than crystalline chloramphneicol. A suspension of the palmitate prepared for pediatric use contained 125 mg. of chloramphenicol per 4 ml. (1 teaspoon). Seventeen patients varying in age from 11 days to 9 years were given 50 mg. per kilogram of body weight per day in four to six equal doses and continued from three to seven days. The parents of these patients reported that the chloramphenicol palmitate suspension was taken without difficulty and in most cases with pleasure. No instances of nausea, vomiting, diarrhea, drug fever, or drug eruption were noted in this small series, and the therapeutic results were considered satisfactory.

HANSEN, Galveston, Texas.

PROTEIN-FREE AND PURIFIED AMNIOTIC FLUID AS AN EQUIVALENT OF BIOLOGICAL ULTRAFILTRATE: ITS THERAPEUTIC POSSIBILITIES. J. ANDRÉ THOMAS, P. PAUL THIERY, and JUDITH BLASS, Etudes neo-natales **1**:103 (Dec.) 1952.

Amniotic fluid of cows and ewes, purified and deproteinized, loses its original biological specificity; it is rich in free amino acids, in reducing sugar (fructose), in mineral elements, in microelements, poor in urea, and slightly alkaline. The mineral and organic constituents are in a state of equilibrium, producing a very remarkable ionic balance.

The amniotic fluids are richer than the blood serums in threonine, cystine, aspartic acid, glutamic acid, and alanine. Injected, they are perfectly tolerated and very rapidly resorbed. They are useful by virtue of their cell-rehydrating properties; since they are equivalent to ultrafiltrates of serum, their use has enabled the authors to obtain massive cultures of certain viruses (particularly that of aphthous fever) on fragments of living skin.

FROM THE AUTHORS' SUMMARY.

PEDIATRIC CONCEPTIONS OF A CENTURY AGO. M. LATO, Clin. pediat. **32**:145 ,(March) 1950.

In a review of an Italian medical book by Gherardo Strucchi published in 1850, entitled "On the Nature, Cause, and Treatment of Rickets, Scrofula, and Pulmonary Phthisis," Lato brings to light the changes in the conceptions of these diseases after 100 years.

HIGGINS, Boston.

News and Comment

GENERAL NEWS

Specialists Certified by American Board of Pediatrics, Inc.—The following physicians were certified as specialists by the American Board of Pediatrics, Inc., after examinations held in Philadelphia, May 1, 2, and 3, 1953:

Allison, Joseph, Waltham, Mass.
Anderson, Herman Weatherby, Fort Worth, Texas.
Anderson, Richard B., Madison, Wis.
Bain, Mary Daly, Methuen, Mass.
Barba, William P., II, Philadelphia.
Baum, Samuel, Passaic, N. J.
Brooks, Clifton Rowland, Newark, Del.
Bruser, David, San Leandro, Calif.
Coffin, S. Farnum, Brookline, Mass.
Cohen, Frank, Fort Worth, Texas.
Connor, Harold L., San Francisco.
Coriell, Lewis L., Camden, N. J.
Cox, Harry Duffield, Portsmouth, Va.
Crothers, Charles H., New Britain, Conn.
Crowell, William Miller, Waverly, Mass.
Curtis, Bernard M., Wantagh, L. I., N. Y.
Deane, Garrett E., Catonsville, Md.
de Lange, Samuel, New York.
Dreifuss, Richard Louis, Forest Hills, L. I., N. Y.
Eldredge, L. L. Jr., Hingham, Mass.
Emerson, Bettina Meyerhof, Newtonville, Mass.
Feldman, Hobart T., Allentown, Pa.
Fifer, John Sherwood, Fort Lauderdale, Fla.
Frame, Eugene Martin, Jacksonville, Fla.
Frazier, Robert Gregory, New York.
Giannini, Margaret Joan, New York.
Greenblatt, Harold J., New Britain, Conn.
Houston, Gilbert, East Greenwich, R. I.
Izzo, Gerard N., Brooklyn.
Johnson, Alvis F., Dallas, Texas.
Kalbacher, Joseph Edward, Westfield, N. J.
Kasales, Clarence J., Flourtown, Pa.
Katz, Selig H., Levittown, L. I., N. Y.
Kellogg, Frank W., Morenci, Mich.
Kellogg, Huston Glenn, San Diego, Calif.
Kibrick, Sidney, Boston.
Kueffner, William Robert, Fairfield, Conn.
Kunz, Hans W., Bronxville, N. Y.
Kurtz, Charles Hertzler, Lancaster, Pa.
Laupus, William Edward, Detroit.
Lewis, S. Robert, Princeton, N. J.
McAllister, Robert M., Camden, N. J.
McClave, Charles R., Columbus, Ohio.
McEvoy, Robert J., Euclid, Ohio.
Mackell, James Valentine, Philadelphia.
Mark, Mortimer L., New York.
Maroney, Margaret, San Rafael, Calif.
Mast, Joseph E., Elizabeth, N. J.
Mastrota, Francis Michael, Washington, D. C.
Mermann, Alan C., Lancaster, Pa.
Mills, Willard Brain, Greenville, S. C.
Moss, Armand H., Brooklyn.
Mullinger, Margaret Adele, Philadephia.
Nesbitt, Robert D., Chicago.
Ostlund, Elvira, Rye, N. Y.
Pitkin, Olive Emma, New York.
Polhemus, Donald W., Pomona, Calif.
Richman, Estelle Melman, Philadelphia.
Rizk, Edward G., Youngstown, Ohio.
Rogers, Kenneth Dyer, Pittsburgh.
Rohrer, Max Leslie, Euclid, Ohio.
Rosenberg, Morton, (MC), U. S. A. F.
Rosenberg, Philip, Philadlephia.
Roth, Robert Otto, McKenney, Va.
Rubenstein, Ira Saul, (MC), U. S. A. F.

Sartwell, Mary K. L., Washington, D. C.
Sato, George, (MC), U. S. A. F.
Schnitzker, William Frederic, Washington, D. C.
Sewell, Edward M., Philadelphia.
Smith, Edward A., Jr., San Francisco.
Smith, Jeanne Helena, New York.
Smith, Nathan James, Philadephia.
Southard, Samuel C., Ventnor City, N. J.
Spencer, William Price, Richmond, Va.
Stepan, Charles Edward, Chicago.
Stricker, Fred Leslie, Denver.
Teate, Hentz Luten, Jr., Decatur, Ga.
Trossman, Chester Marshall, Quonset, R. I.
Ulstrom, Robert Alger, Minneapolis.
Walker, John Samuel, Atlanta, Ga.
Wasserman, Edward, New York.
Wehrle, Paul F., Pittsburgh.
Weil, Marvin Lee, Cincinnati.
Whitlock, Coleman M., Jr., Concord, N. C.
Whitman, Mark Allan, Philadelphia.
Winn, James Sabey, New Brunswick, N. J.
Young, Richard Atlee, Hagerstown, Md.

Pediatricians Certified in Pediatric Allergy by the American Board of Pediatrics, Inc.—The following pediatricians were certified in the subspecialty of pediatric allergy at the examination held in Chicago on April 28, 1953:

Percy Hall Jennings, Jr., Berkeley, Calif.
Maurice Kaufman, Lexington, Ky.
Carroll M. Pounders, Oklahoma City.
Walker L. Rucks, Memphis.

ANNOUNCEMENTS

National Foundation for Infantile Paralysis.—The National Foundation for Infantile Paralysis makes the following statement upon the recommendation of its Advisory Committees on Research and Education:

"The Office of Defense Mobilization, a governmental agency, has been designated the allocating authority for the nation's entire supply of gamma globulin. Inasmuch as this blood fraction is effective in preventing measles, infectious hepatitis and poliomyelitis, and because this substance is in very limited supply, allocation of the nation's stockpile through a central agency was decided upon as the most effective way to prevent the greatest number of cases of these diseases.

"We have just learned that the Office of Defense Mobilization has announced its plan for allocating gamma globulin for use against poliomyelitis. The basis for the plan as announced was recommended by a special panel appointed by the National Research Council, a quasi-governmental agency.

"We note with some concern that in accordance with this plan, the greater part of the nation's stockpile of this scarce material may be used in a manner for which direct proof of efficacy is lacking. Reference is made here to the recommendation that gamma globulin be administered to household and other intimate contacts of patients suffering from poliomyelitis and, in certain circumstances, even to contacts of individuals suspected of having poliomyelitis.

"The field trials conducted during the summers of 1951 and 1952, with financial support of the National Foundation for Infantile Paralysis, demonstrated that gamma globulin, when administered during an epidemic of poliomyelitis to individuals in those age groups subject to greatest risk, provides some temporary protection against the paralytic form of this disease. Whether or not gamma globulin will be equally effective when used in some other manner is unknown. The field trials further provided suggestive evidence that those individuals who develop poliomyelitis following administration of gamma globulin develop a less severe form of the disease.

"While it is true that in a population group made up entirely of individuals who are contacts of persons with poliomyelitis, there develops subsequently an unusually large number of cases of

this disease, it is also true that approximately 75 per cent of these 'secondary' cases occur within six days of the time the first case in the family has been diagnosed. Whether or not gamma globulin will prevent poliomyelitis when it is administered to contacts of diagnosed cases is unknown.

"We know that gamma globulin is not effective if administered to patients after signs of the disease are apparent. We also know that most, if not all, contacts of cases of poliomyelitis are already infected with the virus by the time the first case in the family has been diagnosed. In fact, there is good reason to believe that such individuals might have been infected for a period of several days. If the blood fraction is to be administered to contacts of cases, the important and as yet unanswered question is 'Has the disease already advanced beyond the point where gamma globulin can prevent paralysis?' There is good reason to believe that there will be many contacts of cases of poliomyelitis who will become paralyzed even though they receive gamma globulin.

"The National Foundation's Advisory Committees on Research and Education have heretofore recommended and continue strongly to recommend that the major portion of the nation's stockpile of gamma globulin, available for use in poliomyelitis, be reserved for mass injections of children in the most severe polio epidemics of 1953. This recommendation is made because it is the only method so far proven to be effective against poliomyelitis. The Advisory Committees recommend further that the smaller portion of this scarce material be used in conjunction with contacts of diagnosed cases of poliomyelitis. It is hoped that, in connection with this latter use, adequate studies may be carried out so that we may learn whether or not gamma globulin employed in this manner can prevent paralytic poliomyelitis effectively."

The National Foundation stands ready to assist State Health Officers who may elect to employ community-wide injections of gamma globulin in an effort to halt an epidemic of poliomyelitis. It is prepared to provide specially trained personnel and such items of equipment as would not ordinarily be available in many communities.

American Pediatric Society.—The following are the officers of the American Pediatric Society elected to serve for the year 1953-1954: President, Dr. Rustin McIntosh; Vice-President, Dr. Wilburt C. Davison; Secretary-Treasurer, Dr. Aims C. McGuinness; Recorder-Editor, Dr. Stewart H. Clifford; Member of the Council (7 years), Dr. John A. Anderson. The following candidates were elected to membership: Dr. Neva M. Abelson, Philadelphia; Dr. Janet S. Baldwin, New York; Dr. William Berenberg, Boston; Dr. A. L. Chute, Toronto, Canada; Dr. Clifford G. Grulee, Jr., New Orleans; Dr. Janet Hardy, Baltimore; Dr. Chris P. Katsampes, Rochester, N. Y.; Dr. John D. Keith, Toronto, Canada; Dr. John A. Lichty, Denver; Dr. Edmund R. McCluskey, Pittsburgh; Dr. Robert J. McKay, Burlington, Vt.; Dr. Lawrence B. Slobody, New York; Dr. Alexander J. Steigman, Louisville; Dr. Lewis Thomas, Minneapolis, and Dr. Richard Wagner, Boston.

Society for Pediatric Research.—The Society for Pediatric Research held its 23rd annual meeting in Atlantic City, N. J., on May 4 to 6, 1953. Dr. Robert Ward, New York, presided. The following officers were elected for the year 1954: President, Dr. Harold Harrison, Baltimore; Vice-President, Dr. Russell Blattner, Houston, Texas; Secretary-Treasurer, Dr. Sydney S. Gellis, Boston; members of Council, Dr. Charles Lowe, Buffalo; Dr. C. Henry Kempe, San Francisco.

Dr. Rene Dubos of New York was the guest speaker at the banquet.

Seventh International Pediatric Congress.—The Seventh International Pediatric Congress is to be held at Habana, Cuba, Oct. 12 to 17, inclusive. This will include six plenary sessions, 14 round-tables, free subject sessions, and a large scientific exhibit. There will be presentations by foremost European and North and South American authorities and an attractive entertainment program. For reservations and further details, write to Organizing Committee of International Pediatric Congress, Hospital Mercedes, Vedado, Habana, or to Gomez Tours, Habana.

Postgraduate Course in Pediatric Allergy.—The New York Medical College, 106th and Fifth Aves., New York 29, will give a postgraduate course in pediatric allergy under the direction of Dr. Bret Ratner. The course will extend from Nov. 4, 1953, to May 31, 1954, 30 sessions, on Wednesday, 9 a. m. to 4 p. m., fee $300. Applications should be made to the dean.

PERSONAL

Appointment for Dr. Alan Ross.—Dr. Alan Ross has been appointed professor of paediatrics, McGill University of Medicine and physician-in-chief, The Children's Memorial Hospital, Montreal, Canada, as of July 1st, 1953.

Appointment for Dr. Alton Goldbloom.—Dr. Alton Goldbloom has been named professor emeritus of paediatrics at McGill University of Medicine and consulting physician at The Children's Memorial Hospital, Montreal, Canada.

Books

Biological Antagonism: The Theory of Biological Relativity. By Gustav J. Martin, Sc.D., Research Director, The National Drug Co., Philadelphia, Pa. Price, $8.50. Pp. 516, with 64 figures and 44 tables. The Blakiston Company, 1012 Walnut St., Philadelphia 5, 1951.

This well-written book contains an excellent bibliography on a concept which has become very important in many branches of medicine. The concept of the biological antagonism of chemical analogues is exemplified by the mutual antagonism of *p*-aminobenzoic acid and sulfonamides with reference to the growth of micro-organisms. Those chemical analogues would have been expected to act in vivo either indifferently of one another or synergistically, on the basis of similarity of chemical properties in vitro; the fact is to the contrary the activity of either one with respect to a third chemical complex is depressed by the other. The mode of action has been guessed to be one in which there is competition for a place on an apoenzyme, and some evidence in certain cases is consistent with this view. However, there is no rigorous integration of facts which would justify the use of the term "theory;" predictability is still in the realm of fairly vague generalities. Consequently, the author's claim of a theory of biological relativity is premature. The inclusion of the antibody-antigen reaction of proteins is not well taken, for the reaction does not meet the same criteria as for the example cited and may lead to confusion.

Clinical Electrocardiography. By Captain Ashton Graybiel (MC), U.S.N., Director of Research, U. S. Naval School of Aviation Medicine, Pensacola, Fla. Price, $5. Thos. Nelson & Sons, 19 E. 47th St., New York 17, 1951.

This monograph is intended for the medical practitioner who desires a working knowledge of clinical electrocardiography. About one-third of the book is devoted to the meaning of the electrocardiogram and the electrocardiographic pattern in healthy persons. Brief sections are devoted to the arrhythmias, ventricular hypertrophy, congenital and rheumatic heart disease, infectious diseases, pericarditis, pulmonary heart disease, hypertensive heart disease, and coronary heart disease.

This concise well-written volume will prove of value to the pediatrician who wishes to familiarize himself with the essentials of electrocardiographic diagnosis.

From a Doctor's Heart. By Eugene F. Snyder, M.D. Price, $3.75. Pp. 251, with 16 illustrations. Philosophical Library, Inc., 15 E. 40th St., New York 16, 1951.

Here is a book, autobiographical in nature, which portrays the experience of a physician who has been stricken down in middle life by a crippling disease (coronary thrombosis), and who tells us how he met this catastrophic episode in his life. The foreword is by Dr. Paul Dudley White. The author, with a diverting sense of humor and an awareness of a philosophical approach towards his incapacity, has interestingly described in considerable detail his feelings, his fears, his hopes, his faith, and his resignation to a Higher Power. There is much here for reflection which should provide guidance to those similarly stricken and be a bulwark of strength to those who become victims of panic. As he implies, the latter is often helped and undergirded by a deep religious faith during times of stress.

There is a fine family relationship portrayed between wife (herself a physician) and son and patient, showing how each makes his significant contribution to the author's recovery. Having lived part of their lives in Russia and in Czechoslovakia, and now having settled in a small community in New England, there are international references which extol American democracy and the free world. In our present day these are significant, and should be taken to heart. In these passages the author gives the reader a realization of the good fortune of American citizenship. He extols the general practitioner, his great contribution to medicine, and the important role he plays in good medical care, with stress on the family-doctor relationship. There is a good chapter on body and mind. The author here describes the importance of psychosomatic relationships in disease, and how he, in his own illness, discovered what they meant to him and how they aided him towards recovery. The inference is that emotional disturb-

ances and intrapsychic conflicts, when seen in the young child and adolescent, may lead to hypertension and certain functional cardiac disorders. It is here that psychiatry may contribute much.

To all those who are interested in their heart (and who is not?) this book should be read.

Physical Medicine and Rehabilitation for the Clinician. By Frank H. Krusen, M.D. Price, $6.50. Pp. 371, with 96 figures and 13 tables. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1951.

This textbook is an interesting and informative discussion of the latest developments in all phases of a new and important branch of medicine, physical medicine and rehabilitation. The editor has developed the book from a series of lectures prepared by 24 authors representing six leading university centers of physical medicine and rehabilitation in the United States.

The book is composed of four sections. The first section contains an excellent discussion of the essentials of proper prescription writing for physical therapy and occupational therapy. Available types of treatment are presented with their indications, contraindications, and illustrative prescriptions. The physiologic effects, applications, and uses of heat, massage, and exercise, together with types of training in posture, rest, and relaxation, are thoroughly discussed. Two chapters on occupational therapy and descriptions of ultraviolet radiation, microwaves, and ultrasonic energy complete this section.

In the second section there is a dissertation on electrodiagnostic methods and testing procedures for the evaluation of pain. Skin resistance testing in neurologic diagnosis and measurements of temperature in studies of the circulation are included. This section will be of particular interest to the neurologist.

Section three pertains to the clinical aspects of physical medicine and rehabilitation. It is in this section that the book will have its most universal appeal. Presentations by outstanding authorities cover the physical treatment and rehabilitation of patients with hemiplegia, poliomyelitis, thoracic lesions, and neurologic disorders. Other valuable discussions include such varied conditions as backache, painful feet, arthritis, paraplegia, cerebral palsy, and painful shoulder.

The fourth section is outstanding for its excellent chapters on the fundamentals of anatomy, physiology, and therapeutic exercise and the physiologic effects of rest and immobilization.

This book is a unique presentation of valuable information in a new and essential field of medicine. It is detailed and explicit, with well-chosen bibliographies and good illustrations. It will provide an excellent reference for the physiatrist, the neurologist, the internist, the orthopedist, and the general practitioner.

Brain Tumors of Childhood. By Henry M. Cuneo, Asst. Clinical Professor of Neurological Surgery and Carl W. Rand, Clinical Professor of Neurological Surgery, University of Southern California. Price, $5.75. Pp. 224, with 64 figures. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1952.

This little monograph presents in a clear and concise manner the authors' experiences with intracranial tumors in 83 children. Their observations have been similar to those of others. They found the astrocytomas, particularly those of the cerebellum, to be the most frequent and the most favorable of all the tumors which develop in the brains of children. The patients with medulloblastomas did poorly, and in several instances they, too, found that the tumor had spread to other parts of the central nervous system through the cerebrospinal fluid. They report two unusual cases in this group. In one patient the tumor was not found when the cerebellum was exposed at operation, but she recovered without x-ray therapy and remained practically free from symptoms for six years. Signs of involvement of the thoracic spinal cord then developed, and a laminectomy disclosed a medulloblastoma at that level. In still another case of spinal metastasis of a medulloblastoma the tumor caused erosion of four neighboring thoracic vertebrae. These authors like other surgeons have found that the craniopharyngiomas, ependymomas, pinealomas and papillomas of the choroid plexus present difficult and discouraging surgical problems. In their chapter on "Glioblastoma (Spongioblastoma) Multiforme" they have discussed both that particular type of tumor and the polare spongioblastomas as well. The result is certainly confusing. Their statement that, "Fortunately the spongioblastoma seldom occurs in children," is unfortunately true only for the glioblastoma multiforme. It was not Globus who designated this tumor a "glioblastoma multiforme;" that was the name applied by Bailey.

Globus called it a "spongioblastoma multiforme," and for a time Bailey followed his lead. Meningiomas, gliomas of the optic nerve, metastatic and malignant tumors, eosinophilic granulomas of the skull, and infectious granulomas of the brain are also well presented.

This is a sober conservative presentation of the subject which students, pediatricians, neurologists, and neurological surgeons will find accurate and helpful. The text is clear. The figures are well chosen and well reproduced.

Allergy in Relation to Pediatrics. By B. Ratner. Price, $3.75. Pp. 228, with 10 illustrations. Bruce Publishing Company, 2642 University Ave., St. Paul 4, 1951.

This is the most scholarly book on allergy in pediatrics that is available at this time. Dr. Bret Ratner has chosen 16 authorities and experts on this subject. They have had extensive practical experience in pediatrics and have pioneered pediatric allergy. The text is practical and should be readily understood by the average pediatrician. He needs this information to apply in his daily contact with infants and children. Although there is some duplication and overlapping of subjects, such as description of symptoms, skin testing, and treatment of various allergic manifestations, this only serves to emphasize the importance of these subjects. The very few charts and graphs make this text delightful to read and easy to follow. Pediatricians may not be willing to accept some of the suggested implications of the psychodynamics of allergic disease. This same type of reasoning could apply to other chronic diseases of children as one of a number of factors that aggravate or exaggerate symptoms of these illnesses without being a direct etiological factor.

The last chapter summarizes the entire subject in a most common sense manner. Allergy is a vital part of our everyday practice, and probably the most neglected because it is the least understood. "Allergy in Relation to Pediatrics" is a worthwhile and necessary contribution to pediatric literature. Every pediatrician should not only read this book but use it frequently as a reference text.

Dr. Ratner has produced a vital baby and it is hoped that he will father more volumes relating to "Allergy in Pediatrics."

A Guide to Children's Dosage. By A. M. Earle. Price, not given. Pp. 78. University of Arkansas, Little Rock, Ark., 1951.

This small pocket volume was compiled as an aid to students and interns at the University of Arkansas School of Medicine. It follows in part the recommendations of Poncher and Numa and associates regarding dosage of certain drugs. For the most part calculation is based on weight and is according to accepted methods. Differences between oral, parenteral, and rectal dosages are indicated. The list of drugs included is by no means complete, but except for one or two omissions is adequate. Ephedrine is missing. On the other hand, all of the antibiotics and corticotropin (ACTH) and cortisone are included.

This is a useful little book and should be helpful for students and interns.

La Torsion tibiale: Sa mesure; Son intérêt, clinique, radiologique et chirurgical. By P. Dupuis. Paper. Price, 920 francs. Pp. 196, with 33 illustrations and 8 tables. Masson & Cie, 120 Boulevard Saint-Germain, Paris, 6e, 1951.

This book is dedicated to Professor Louis Ombredanne, who was one of the first to propose research into the etiology of tibial torsion; and to Professor Le Damany, who carried out many of the studies reported in this book.

Tibial torsion is distinctly different from tibial rotation. The condition which has been studied and described in this small book has been considered from the standpoint of etiology, anatomical findings, clinical considerations, and treatment. Tibial torsion may be produced by imbalance of the muscles which control the movements of the foot in inversion or eversion. It is always present in uncorrected congenital talipes equinovarus, and may be the result of long-standing paralysis from poliomyelitis or cerebrospastic inversion or eversion of the foot. Perhaps the most common cause of tibial torsion is contracture and shortening of the iliotibial band following poliomyelitis. Other conditions mentioned, but rarely observed today on the North American continent, would include rickets or the bone dystrophies of unknown etiology. Treatment of tibial torsion is discussed in detail and an exceptionally complete bibliography of the entire subject is appended.

Diabetes Guide Book for the Physician. Prepared by the committee on education of the American Diabetes Association, Inc. Gratis. Pp. 79. New York: The Association, 1950.

This book is prepared by the committee on education of the American Diabetes Association. It is pocket size and is a guide for physicians. Diagnosis and treatment of diabetes mellitus, hypoglycemic reactions, and diabetic coma are outlined. Diabetic diets and food exchanges with a few sample meals are listed. Very little time is devoted to the management of diabetes in children. This guide book should be of help to physicians and, likewise, of value to patients.

Estreptomicina y nuevos antibioticos en la terapeutica infantil. By Jose M. Albores. Pp. 401, with 75 tables and graphs. Paper. No price given. Printed in Argentina. Editorial Universitaria Buenos Aires, Buenos Aires, 1951.

This book is a thorough analytical and critical study of the action and use of antibiotics in pediatrics. The first eight chapters deal with streptomycin and include the composition, production and action of the antibiotic, as well as the experimental studies, absorption, action, intolerance, and method of administration. An entire chapter is devoted to its application in the various phases of tuberculosis. To this is added a most thorough discussion of the various manifestations of tuberculosis with adjuvant therapeutic agents and the treatment of allied conditions that are nontuberculous.

Final chapters are devoted to aureomycin, chloromycetin, oxytetracycline ("terramycin"), polymixin, bacitracin, "garlicina," and "allicin." These are all very thoroughly discussed in the same manner as streptomycin. It is difficult to evaluate the true excellency of this work. The time taken to assemble the immense and complete biography must have been tremendous. Although written in Spanish this is by far the most readable and complete work that has appeared on this varied subject. It contains all that is necessary to understand the considerable study that has come into the medical world in the last few years on this very vital subject. It is recommended to every class of readers.

Rehabilitation Nursing. By Alice B. Morrissey. Price, $5. Pp. 285, with 58 illustrations. G. P. Putnam's Sons, 210 Madison Ave., New York 16, 1951.

"The ultimate aim of rehabilitation is to restore the handicapped individual to the fullest physical, mental, social, vocational, and economic usefulness of which he is capable."

The above statement in the preface of the book gives the aims of the author and has been brought out in a simple, straightforward way to make it valuable for the student nurse, visiting nurse and all phases of nursing.

The nurse's responsibility in maintaining physical and mental health during rehabilitation and in teaching the patient how to maintain good health after his return to the community is paramount.

"Cognizant of the ultimate complete rehabilitation of her patient, the nurse is obligated to acquire thorough understanding of posture techniques and she must develop the ability to apply the principles of good body alignment and correct positioning of the patient in all areas of nursing care."

A brief historical summary of rehabilitation and more detailed present-day procedures with many references for further study make it a valuable text and reference book.

Angiocardiography As A Diagnostic Aid In Congenital Heart Disease. By J. W. C. DeGroot, M.D. Price, not given. Pp. 108, with 20 illustrations. Keesing, Amsterdam-Z, Ruysdaelstraat 71, Amsterdam, Holland, 1951.

The first portion of this monograph is devoted to a review of the work of other investigators in the field of angiocardiography.

The second portion records the author's own experience, which was limited to a total of 51 angiocardiograms. Out of this group he discusses in detail the findings in 13 cases consisting of various congenital malformations of the heart.

The book is of value to the reader who wishes a brief introduction to the methods and limitations of angiocardiography as a diagnostic aid in congenital heart disease.

Directory of Pediatric Societies *

INTERNATIONAL

International Union for Child Welfare

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 16 rue du Mont Blanc, Geneva, Switzerland

IV° Congreso Pan-Americano de Pediatria

President: Dr. Leonel Gonzaga.
General Secretary: Dr. Adamastor Barbosa.
Secretariat: Rua Dias da Rocha 25, ap. 602, Copacabana, Rio de Janeiro.
Place. Rio de Janeiro. Time: 1954.

IV° Congreso Sudamericano de Pediatria, Confederacion Sudamericano de Sociedades de Pediatria

President: Prof. Carlos Prado.
Vice-President: Dr. Marcelo Garcia.
General Secretary: Dr. Alvaro Aguiar.
Place: Avenida Mem de Sá 197, Rio de Janeiro. Time: 1954.

FOREIGN

Academy of Medicine, Toronto, Section of Paediatrics

Chairman: Dr. Bernard Laski, Toronto.
Secretary: Dr. Crawford Anglin, Toronto.
Place: Academy of Medicine.

Australian Paediatric Association

President: Dr. Robert Southby.
Secretary: Dr. J. Colebatch, 12 Collins St., Melbourne.
Place: Canberra. Time: Yearly in April.

British Paediatric Association

President: Prof. J. Smellie.
Secretary: Prof. Alan Moncrieff, Institute of Child Health, The Hospital for Sick Children, Great Ormond St., W. C. 1, London.
Place: Windermere. Time: Between Easter and Whitsunday.

Canadian Paediatric Society

President: Dr. D. B. Leitch, 210 McLeod Bldg., Edmonton, Al.
Secretary: Dr. R. L. Denton, 1615 Cedar Ave., Montreal 25.

Danish Pediatric Society

President: Dr. A. Rothe-Meyer, Children's Hospital Fûglebakken, Copenhagen F.
Secretary: Dr. E. Winge Flensborg, Children's Hospital, Martinsvej, Copenhagen V.
Place: Different hospitals. Time: First Wednesday of every month.

Departmento de Pediatria da Associação Paulista de Medicina

President: Dr. Oswaldo Riedel de Souza e Silva.
Secretaries: Dr. Ovidio Collesi.
Dr. Helda Elina Vieira de Barros.
Place: Avenida Brigadeiro Luiz Antonio, 278, São Paulo, Brasil.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Deutsche Gesellschaft für Kinderheilkunde

President: Prof. Dr. Adam, Universitats Kinderklinik, Erlangen.
Secretary: Prof. Dr. Jochims, Städtische Kinderklinik, Lübeck.

Hellenic Society of Pediatrics

President: Dr. Spiros Charocopos, Alexandras Ave. 1, Athens.
Secretary: Dr. Nikas Zervos.
Place: Amphitheater, Medical School of the University of Athens. Time: Once a month

Nederlandse Vereeniging voor Kindergeneeskunde

President: Dr. G. J. Huët, 39 Brediusweg, Bussum.
Secretary: Dr. H. Reerink, % Sophia Kinderziekenhuis, 160 Gordelweg, Rotterdam.
Place: Children's Hospital or Pediatric Clinic. Time: Three times a year.

Norsk Pediatrisk Selskap

President: Dr. Arne Njå, Sporveisgt. 29, Oslo.
Secretary: Dr. Helge Granrud, Abbedikollen 38, Bestun, Oslo.
Place: Barneklinikken, Rikshospitalet, Oslo. Time: Friday, six times a year.

Pediatric Society of New Zealand

President: Dr. Edward J. Cronin, Power Board Bldg., Queen St., Auckland.
Secretary: Dr. J. D. Matthews, 104 Symonds St., Auckland.

Philippine Pediatric Society

President: Dr. Renato Ma. Guerrero.
Secretary: Dr. Felix A. Estrada.
Place: Santo Tomas University Hospital and other pediatric centers. Time: Every two months.

Royal Society of Medicine, Section of Paediatrics

President: Dr. D. W. Winnicott.
Secretary: Dr. J. J. Kempton; Dr. A. P. Norman.
Sectional Editor: Dr. D. MacCarthy
Place: 1 Wimpole St., London W.1. Time: Fourth Friday of each month, 4:15 p. m.

Schweizerische Gesellschaft für Pädiatrie

President: P. D. Dr. F. Bamatter, St.-Léger 8, Genève.
Secretary: Dr. M. Nicod, Place Bel-Air 2, Lausanne.

Sociedad Argentinade Pediatrie

President: Dr. Rudolfo Kreutzer.
Secretary: Dr. Luis M. Cucullu.
Place: Sante Fè 1178, Buenos Aires.

Sociedad Boliviano de Pediatria

President: Dr. Carlos Daza Meruvia.
Secretary: Dr. Cecilio Abela Deheza.
Place: S. C. I. S. P. Bldg. Time: Every Thursday.

Sociedade Brasiliera de Pediatria

For officers, see IV° Congreso Sudamericano de Pediatria, above.

Sociedad Chihuahuense de Pediatría

President: Dr. Alfonso Ruiz Escalona.
Secretary: Dr. Armando G. Herrera.
Place: Hospital Infantil del Estado, Chihuahua, Chih., Mexico.

Sociedad Chilena de Pediatria

President: Prof. Dr. Raúl Gantes A.
Secretary General: Dr. Edmundo Cardemil.
Place: Medical Society, Merced 565, Santiago.

Sociedad Colombiana de Pediatria

President: Dr. Jorge Camacho Gamba.
Vice-President: Dr. Antonio Alvarez Riaño.
Secretary: Dr. Hernando Vanegas A.
Treasurer: Dr. Efrain Perry Villate.

Sociedad Cubana De Pediatria

President: Prof. Agustin Castellanos y Gonzales.
Secretary: Dr. Carlos Hernandez Miyares, Apartado 2430, Habana.
Place: Catedra de Clinica Infantil Hospital Nuestra Senora de las Mercedes and Hospital Municipal de Infancia, Habana. Time: 9 p.m., last Wednesday of every month.

Sociedad de Pediatría de Concepción (Chile)

President: Dr. Próspero Arriagada.
Secretary: Dr. Guillermo Repetto D.
Place: Hospital Clinico Reginal Concepción. Time: 7:30 p.m., second Tuesday of every month, April to December, inclusive.

Sociedad de Pediatria del Litord

President: Dr. Raul Bruera.
Vice-President: Dr. Isidoro Slullitel.
Secretary General: Dr. Elsa Galimany.

Sociedad de Pediatria y Puericultura del Atlantico

President: Dr. Atilio Marino.
Vice-President: Dr. Helena Uribe de Jimenez.
Secretary-Teasurer: Dr. Hernando Borrios Angulo.

Sociedade de Pediatria do Rio Grande do Sul

President: Dr. Francisco Talaia de Moura.
Vice-President: Dr. Breno Barcellos.
Secretary: Dr. Enio Pilla and Dr. Damasceno Tilho.
Treasurer: Dr. J. Sant' Ana Aúde.

Sociedade Portugesa de Pediatria

President: Prof. Dr. Lucio de Almeida.
Vice-President: Dr. Cordeiro Ferreira.
Secretary General: Prof. Dr. Carlos Salazar de Sousa.
Treasurer: Dr. Mario Cordeiro.
Place: Clinica pediatrica do Hospital D. Estefania, Lisbon. Time: Monthly.

Sociedad Uruguaya de Pediatria

President: Dr. Euclides Peluffo.
Secretary: Dr. Ruben Gorlero Bacigalupi.
Place: Av. Agraciada 1464, piso 13, Montevideo.

Sociedad Venezolana de Puericultura y Pediatría

President: Dr. Guillermo Rangel.
Secretary: Dr. Guillermo Tovar E., P.O. Box 5, Caracas.
Place: Hospital Municipal de Niños, Caracas. Time: Once a month.

SOCIEDAD YUCATECA DE PEDIATRIA

President: Dr. Humberto Perez Concha.
Secretary: Dr. Prudencio Ruiz Salazar.
Treasurer: Dr. Jorge Antiveros Andráde.

LA SOCIÉTÉ DE PÉDIATRIE DE MONTRÉAL

President: Dr. Henri Charbonneau, 3095 Sherbrooke, Montréal.
Secretary: Dr. Edmond Baril, 301 Carré St-Louis, Apt. 1, Montréal.
Time: Second Thursday of each month.

SOCIÉTÉ DE PÉDIATRIE DE PARIS

President: Prof. Jean Cathala, 45 rue Scheffer, Paris XVI.
Secretary: Prof. Maurice Lamy, 94 rue de Varenne, Paris VII.
Place: Hôpital des Enfants Malades, 149 rue de Sèvres, Paris XV. Time: 4 p.m., third Tuesday of each month.

SOCIÉTÉ DE PÉDIATRIE DE QUÉBEC

President: Dr. Roland Thibaudeau, 203 des Erables, Québec.
Secretary: Dr. Euclide Dechêne, 312 du Roi, Québec.
Place: Quebec. Time: May and November.

SOUTH AFRICAN PAEDIATRIC ASSOCIATION

Chairman: Dr. Seymour Heymann, 804 Medical Centre, Jeppe St. Johannesburg.
Honorary Secretary-Treasurer: Dr. I. Kessel, Transvaal Memorial Hospital for Children, Milner Park, Johannesburg.

SWEDISH ASSOCIATION OF PEDIATRICS

President: Prof. Sture Siwe, Lund.
Secretary: Dr. Karl-Axel Melin, Färjestadsvägen 16, Bromma.

SWEDISH MEDICAL SOCIETY, SECTION FOR PEDIATRICS AND SCHOOL HYGIENE

President: Docent Urban Hjarne, Stockholm.
Secretary: Dr. M. d'Avignon, Pediatric Clinic, Karolinska Sjukhuset, Stockholm 60.
Place: Swedish Medical Society, Klara Östra Kyrkogata 10, Stockholm, and Pediatric Clinic, Uppsala. Time: Second Friday of each month, except summer.

UNITED STATES

National

AMERICAN MEDICAL ASSOCIATION, SECTION ON PEDIATRICS

Chairman: Dr. Eugene T. McEnery, 4458 W. Madison St., Chicago 24.
Secretary: Dr. Wyman C. C. Cole, 3001 W. Grand Blvd., Detroit.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Warren W. Quillian, 140 Alhambra Circle, Coral Gables, Miami 34, Fla.
Secretary: Dr. E. H. Christopherson, 636 Church St., Evanston, Ill.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Harold Harrison, Baltimore.
Vice-President: Dr. Russell Blattner, Houston, Texas.
Secretary-Treasurer: Dr. Sydney S. Gellis, Boston.

THE AMERICAN PEDIATRIC SOCIETY

President: Dr. Rustin McIntosh.
Vice-President: Dr. Wilburt C. Davison.
Secretary-Treasurer: Dr. Aims C. McGuinness.

Sectional

Intermountain Pediatric Society

President: Dr. John T. Mason.
Secretary: Dr. Stanley R. Child.
Place: Ambassador Athletic Club, Salt Lake City. Time: 7 p.m.

New England Pediatric Society

President: Dr. Albert W. Fellows.
Secretary: Dr. Harry Shwachman, 300 Longwood Ave., Boston.
Place: Boston, except Spring meeting. Time: Wednesdays, four times a year.

North Pacific Pediatric Society

President: Dr. Carl Ashley, 2455 N. W. Marshall St., Portland, Ore.
Secretary-Treasurer: Dr. S. Gorham Babson, 1107 S. W. 13th St., Portland, Ore.

Northwestern Pediatric Society

President: Dr. F. G. Hedenstrom, 420 Lowry Medical Arts Bldg., St. Paul.
Secretary: Dr. Charles L. Steinberg, 1118 Lowry Medical Arts Bldg., St. Paul.
Place: Minneapolis; St. Paul; Duluth, Minn., and Rochester, Minn. Time: January, April, July, and October.

Rocky Mountain Pediatric Society

President: Dr. John Lichty, Pediatric Department, Colorado General Hospital, 4200 E. 9th Ave., Denver.
Secretary: Dr. Edwin T. Williams, 1850 Gilpin St., Denver 6.
Place: Announced by mail. Time: Monthly dinner meetings, October to June.

Southwestern Pediatric Society

President: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills, Calif.
Secretary: Dr. Carl A. Erickson, 960 E. Green St., Pasadena, Calif.
Place: Jonathan Club of Los Angeles. Time: 6:30 p.m., second Wednesday in January, March, May, September, and November.

State

Alabama Pediatric Society

President: Dr. David B. Monsky, 748 Adams St., Montgomery 5.
Secretary: Dr. Alice H. Pye, 305 Church St., Montgomery.
Place: Montgomery.

Arizona Pediatric Society

President: Dr. Henry E. Running.
Secretary: Dr. William F. Schaffman.
Time: On Call.

California Medical Association, Section on Pediatrics

Chairman: Dr. Joseph W. St. Geme, 511 S. Bonnie Brae, Los Angeles 5.
Secretary: Dr. Alvin H. Jacobs, 2201 Webster St., San Francisco 15.
Assistant Secretary: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills.

Florida State Pediatric Society

President: Dr. Charlotte C. Maguire, 1413 Kuhl Ave., Maguire Bldg., Orlando.
Secretary-Treasurer: Dr. Wesley S. Nock, 273 Alhambra Circle, Coral Gables.

Georgia Pediatric Society

President: Dr. John A. Simpson, Athens.
Secretary: Dr. J. Harry Lange, 490 Peachtree St., Atlanta.
Place: Savannah. Time: At time of annual state meeting.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Joseph Hetzel, 51 Central Ave., Waterbury.
Secretary: Dr. James H. Root Jr., 103 N. Main St., Waterbury.
Time: With the Connecticut State Medical Society in the spring, with the Connecticut State Clinical Congress in the fall; winter meeting usually in December.

Illinois State Medical Society, Section on Pediatrics

Chairman: Howard R. Miller, 418 Jefferson Bldg., Peoria 2.
Secretary: Harry H. Boyle, 2376 E. 71st St., Chicago 49.

Indiana State Pediatric Society

President: Dr. Harold D. Lynch, Evansville.
Secretary: Dr. L. T. Meiks, Indianapolis.

Iowa Pediatric Society

President: Dr. John McKitterick, 315 Tama Bldg., Burlington.
Secretary: Dr. James Dunn, 501 Davenport Bank Bldg., Davenport.

Massachusetts Hospital Association

President: Dr. Philip D. Bonnet, Administrator, Massachusetts Memorial Hospitals, Boston.
Executive Secretary: Henry G. Brickman, 14 Somerset St., Boston 8.

Michigan State Medical Society, Pediatric Section

President: Dr. Horace L. French, 301 Seymour Ave., Lansing.
Secretary: Dr. Park S. Bradshaw, 1014 Jefferson St., Muskegon.

Montana Pediatric Society

President: Dr. Orville M. Moore, 555 Fuller Ave., Helena.
Secretary: Dr. Joseph W. Brinkley, 410 Central Ave., Great Falls.
Place: Variable. Time: Spring and fall.

Nebraska Pediatric Society

President: Dr. Michael Crofoot.
Vice-President: Dr. Donald C. Nilsson.
Secretary-Treasurer: Dr. A. J. Lombardo, 401 Aquila Ct., Omaha.
Place: Children's Memorial Hospital. Time: 6 p.m., dinner, third Thursday of each month.

New Hampshire Pediatric Society

President: Dr. Douglass W. Walker, Laconia.
Secretary: Dr. Wm. D. Penhale, Concord.
Place. Alternately Concord, Hanover, Laconia, and Manchester. Time: Four times a year.

New Mexico Pediatric Society

President: Dr. M. K. Wylder, Suite 14, Medical Arts Sq., Albuquerque.
Secretary: Dr. Ly Werner, Suite 25, Medical Arts Sq., Albuquerque.
Place: Variable. Time: Every two or three months.

North Carolina Pediatric Society

President: Dr. Eugene Keiter, Kinston Clinic, Kinston.
Vice-President: Dr. Donald Robinson, 308 W. Davis St., Burlington.
Secretary-Treasurer: Dr. Robert Pope, Wilson Clinic, Wilson.
Place: Roaring Gap and Chapel Hill. Time: Sept. 12 and Oct. 30, respectively.

South Carolina Pediatric Society

President: Dr. Walter Moore Hart, 117 W. Cheves St., Florence.
Secretary: Dr. John R. Paul Jr., 82 Rutledge Ave., Charleston.
Place: Columbia, S. C. Time: Second Monday and Tuesday in September.

Tennessee Pediatric Society

President: Dr. O. L. Von Canon, 846 McCallie Ave., Chattanooga.
Secretary: Dr. Oliver W. Hill, 4807 Newcom Ave., Knoxville.

Texas Pediatric Society

President: J. A. Bybee, Beaumont.
President-Elect: Dr. Robert L. Moore, Dallas.
Secretary: Dr. M. C. Carlisle, 1410 Austin Ave., Waco.
Treasurer: Dr. Jack M. Woodall, Big Springs.

Virginia Pediatric Society

President: Dr. Edwin L. Kendig Jr.
Secretary: Dr. T. J. Humphries.

Wisconsin State Medical Society

Chairman: Dr. N. L. Low, Racine
Vice-Chairman: Dr. E. H. Pawsat, Fond du Lac.
Secretary: Dr. Samuel E. Kohn, Milwaukee.
Delegate: Dr. K. J. Winters, Wauwatosa.
Alternate Delegate: Dr. F. Mellencamp, Milwaukee.

Local

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. C. T. J. Dodge, 15457 Euclid Ave., Cleveland 12.
Secretary: Dr. Beulah Wells, 10515 Carnegie Ave., Cleveland 6.
Place: Allen Memorial Medical Library. Time: 8:15 p.m., October, December. February, and March.

Baltimore City Medical Society, Pediatric Section

President: Dr. Wilson Grubb, 4 E. 33d St., Baltimore 18.
Secretary: Dr. Gibson J. Wells, 3101 St. Paul St., Baltimore 18.
Place: 1211 Cathedral St., Baltimore 1. Time: 8:30 p.m., second Tuesday in November, January, and March.

Bronx Pediatric Society

President: Dr. M. H. Grand, 1749 Grand Concourse, New York.
Secretary: Dr. Charles T. Fried, 1220 Grand Concourse, New York.
Place: Morrisania City Hospital. Time: 8:30 p. m., second Wednesday of each month, except June, July, August, and September.

Brooklyn Academy of Pediatrics

President: Dr. Stanley S. Lamm, 20 Plaza St., Brooklyn.
Secretary: Dr. Henry Cohen, 1761 E. 19th St., Brooklyn.
Place: Granada Hotel, Brooklyn. Time: 8:45 p.m., fourth Wednesday of February, March, April, October, and November.

Buffalo Pediatric Society

President: Dr. Douglas P. Arnold.
Secretary: Dr. Richard A. Downey.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p.m., first Monday of each month, September to June.

Central New York Pediatric Club

President: Dr. Frank Williams, 58 Swan St. S., Albany.
Secretary: Dr. Fred W. Bush, 325 Park Ave., Rochester.
Place: Various cities in New York State; fall meeting jointly with Upper New York State Academy of Pediatrics. Time: Third Tuesday of April and October.

Central Ohio Pediatric Society

President: Dr. O. W. Hosterman, 350 East Broad St., Columbus 15.
Secretary-Treasurer: Dr. John Riepenhoff, 2998 Sullivant Ave., Columbus.
Place: As announced. Time: Third Wednesday of September, November, January, March, and May.

Chicago Pediatric Society

President: Dr. Alfred S. Traisman.
Vice-President: Dr. Eugene T. McEnery.
Secretary: Dr. Matthew M. Steiner, 6354 N. Broadway.
Treasurer: Dr. Maxwell P. Borovsky.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Cincinnati Pediatric Society

President: Dr. Nicholas M. Kotona, 5771 Glenway Ave., Cincinnati 38.
Secretary: Dr. John E. Allen, 2546 Auburn Ave., Cincinnati 19.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

Cleveland Academy of Medicine, Pediatric Section

Chairman: Dr. Robert Hauver, 10515 Carnegie Ave.
Secretary: Dr. William Herman, 10300 Carnegie Ave.

Corpus Christi Pediatric Society

President: Dr. Edwin A. Danford.
Secretary: Dr. Jack F. McKemie.
Place: As announced. Time: 7:30 p. m., fourth Wednesday of January, March, May, July, September, and November.

Dade County Pediatric Society

President: Dr. Lynn Whelchel, DuPont Bldg., Miami, Fla.
Secretary: Dr. Howard Engle, 311 Lincoln Rd., Miami Beach, Fla.

Dallas Pediatric Society

President: Dr. Floyd A. Norman, 4325 Cole Ave., Dallas.
Secretary-Treasurer: Dr. Charles L. Smith, 4207 Lemmon Ave., Dallas.
Place: Children's Hospital of Texas. Time: 6:30 p. m., first Thursday in each month.

Detroit Pediatric Society

President: Dr. Ruben Meyer, 938 Maccabees Bldg., Detroit.
Secretary: Dr. W. W. Zuelzer, Children's Hospital of Michigan, Detroit.
Place: Variable. Time: Five times a year.

Honolulu County Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu.
Corresponding Secretary and Treasurer: Dr. John H. Peyton, Kauikeolani Children's Hospital, Honolulu.

Honolulu Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu.
Recording Secretary: Dr. M. H. Hasegawa.
Corresponding Secretary: Dr. John Peyton, Kauikeolani Children's Hospital, Honolulu.
Place: At doctors' homes in rotation. Time: 6 p.m., third Thursday of each month.

Houston Pediatric Society

President: Dr. Emanuel B. Brandes, 3101 Fannin St., Houston.
Secretary: Dr. Robert D. Lomas, 5101 Fannin St., Houston.
Place: River Oaks Country Club. Time: 7:30 p.m., fourth Tuesday of each month.

Kansas City Southwest Pediatric Society

President: Dr. Frank Hogue, Plaza Medical Bldg., Kansas City, Mo.
Secretary: Dr. S. J. Kranson, Truman Rd. and Ash, Independence, Mo.
Place: University Club, Kansas City, Mo. Time: On call.

Long Beach Pediatric Society

President: Dr. Kenneth Berkaw, 211 Cherry Ave., Long Beach, Calif.
Secretary: Dr. C. W. Latshaw, 3821 Atlantic Ave., Long Beach, Calif.
Place: Victor Hugo Restaurant, 730 E. Broadway. Time: First Tuesday night of every month.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Sidney Rosin, 6230 Wilshire Blvd., Los Angeles.
Secretary: Dr. Arthur J. Holmes, 65 N. Madison Ave., Pasadena 1.
Place: Los Angeles County Medical Association Bldg., 1025 Wilshire Blvd., Los Angeles. Time: Second Thursday of alternate months beginning in September.

Louisville Pediatric Society

President: Dr. Lee Palmer.
Secretary: Dr. Martin Z. Kaplan.
Place: Variable. Time: Five times a year.

Medical Society of the County of Kings and Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Max Palinsky, 1270 45th St., Brooklyn 19.
Secretary: Dr. Bernard Greenberg, 670 Ocean Ave., Brooklyn 26.
Place: Kings County Medical Society Auditorium, 1313 Bedford Ave., Brooklyn. Time: 9 p.m., fourth Monday of January, March, April, October, and November.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. George Maksim, 1418 Good Hope Rd. S. E., Washington 20.
Secretary: Dr. John E. Cassidy, 5402 Connecticut Ave. N.W., Washington.
Place: Medical Society Bldg., 1718 M St. N.W., Washington. Time: Spring and fall.

Memphis Pediatric Society

President: Dr. James G. Hughes, 20 S. Dunlap, Memphis.
Secretary: Dr. George S. Lovejoy, 609 N. Highland, Memphis.
Place: Rotated among four hospitals. Time: 7:30 p.m., second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. Franklin Mellencamp, 1513 E. Capitol Dr., Milwaukee.
Secretary: Dr. Karl Beck, 1411 Wauwatosa Ave., Milwaukee.
Place: Milwaukee University Club. Time: Second Wednesday of February, April, June, and October.

Mobile Pediatric Society

President: Dr. Daniel Sullivan, 1420 Government St., Mobile, Ala.
Secretary: Dr. Robert O. Harris III, 1514 Government St., Mobile, Ala.
Place: Admiral Semmes Hotel. Time: 7:30 p.m., fourth Thursday of September, November, January, March, and May.

Nashville Pediatric Society

President: Dr. Thomas Weaver, Bennie Dillon Bldg., Nashville, Tenn.
Secretary: Dr. Ethel Walker, 3415 West End Ave., Nashville, Tenn.
Place: Homes of members. Time: Third Thursday of every month.

Nassau Pediatric Society

President: Dr. Daniel Blitz, 1 Club Drive, Woodmere, N. Y.
Secretary-Treasurer: Dr. David J. Posner, 98 Wolcott Rd., Levittown, N. Y.
Place: Garden City Hotel, Garden City, L. I., N. Y. Time: 7:30 p. m., second Monday of October, December, February, April, and June.

New York Academy of Medicine, Section on Pediatrics

Chairman: Dr. Jerome L. Kohn, 50 W. 96th St., New York 25.
Secretary: Dr. Lawrence B. Slobody, 1 E. 105th St., New York 12.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8:30 p.m., second Thursday of each month, November to May, inclusive.

Northern California Pediatric Society

President: Dr. John A. Anderson, Stanford Hospital, 2351 Clay St., San Francisco 15.
Secretary: Dr. Edward Campion, 1018 E St., San Rafael.
Place: San Francisco. Time: Quarterly, as speakers are available.

Oklahoma City Pediatric Society

President: Dr. C. M. Bielstein, 301 N.W. 12th St., Oklahoma City.
Secretary: Dr. Louis Frank, 1200 N. Walker St., Oklahoma City.
Place: Oklahoma Club. Time: On call.

Philadelphia Pediatric Society

President: Dr. John B. Bartram, 2600 Lawrence St., Philadelphia 33.
Secretary: Dr. F. H. Harvie, 133 S. 36th St., Philadelphia 4.
Place: College of Physicians, 19 S. 22d St. Time: 8:30 p.m., second Tuesday from October to May, inclusive.

Pittsburgh Pediatric Society

President: Dr. Christian John Stoecklein, 519 Medical Arts Bldg., Pittsburgh 13.
Secretary-Treasurer: Dr. James W. Stirling, 694 Lincoln Ave., Pittsburgh 2.
Place: Western State Psychiatric Institute and Clinic. Time: Second Friday in October, December, February, and April.

Queens Pediatric Society

President: Dr. Samuel Dillon, 119-20 Rockaway Beach Blvd., Rockaway Park, N. Y.
Secretary: Dr. Moe Goldstein, 111-32 76th Ave., Forest Hills, N. Y.
Place: Seminole Club, 112th St. and 70th Rd., Forest Hills, N. Y. Time: Second Tuesday in October, December, February, and April.

Richmond Pediatric Society

President: Dr. Hobart T. Dougan, 1832 Monument Ave., Richmond 20, Va.
Secretary: Dr. Robert A. Hoffman, 1001 W. Franklin St., Richmond 20, Va.
Place: Medical College Hospital. Time: 8 p.m., third Thursday of each month, except June, July, and August.

San Antonio Pediatric Society

President: Dr. Edith M. Bonnet, 228 Medical Arts Bldg., San Antonio 5.
Secretary: Dr. Virginia S. Stovall, 1116 S. Texas Bldg., San Antonio 5.
Time: 7:30 p. m., supper, first Tuesday of each month.

Seattle Pediatric Society

President: Dr. O. William Anderson.
President-Elect: Dr. Robert Tidwell.
Secretary-Treasurer: Dr. James L. Tucker.
Place: College Club. Time: 6:30 p.m., fourth Friday of each month from September to June.

St. Louis Pediatric Society

President: Dr. Maurice J. Lonsway.
Secretary: Dr. John C. Herweg.
Place: Candlelight House, 7800 Clayton Rd. Time: 6:30 p.m., second Thursday of each month from October to May, inclusive.

Santa Barbara Pediatric Society

President: Dr. Clarence B. Hills, 22B. W. Micheltorena St., Santa Barbara, Calif.
Secretary: Dr. Louise P. Brown, 3017 Foothill Rd., Santa Barbara, Calif.
Place: Varies. Time: Two to three times yearly, on call.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Fred J. Chapin, 101 John St., Bay City.
Secretary-Treasurer: Dr. George H. Lowrey, University Hospital, Ann Arbor.
Place: University Hospital, Ann Arbor.

Waco Pediatric Society

President: Dr. J. M. Garrett, 1408 Austin Ave., Waco, Texas.
Secretary: Dr. C. G. Shellenberger, 1318 Austin Ave., Waco, Texas.
Place: 1917 Austin Ave., Waco, Texas. Time: Third Tuesday of each month.

Westchester County Medical Society, Pediatric Section

President: Dr. Herbert Kahan, 650 Main St., New Rochelle, N. Y.
Secretary: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: The Roger Smith Hotel, White Plains, N. Y. Time: Four dinner meetings a year, November, February, April, and June.

Western Michigan Pediatric Society

President: Dr. A. Morgan Hill, 310 Fulton, E., Grand Rapids.
Secretary: Dr. John C. Montgomery, 1810 Wealthy St., S.E., Grand Rapids.
Place: Two meetings in Grand Rapids, two elsewhere. Time: First Wednesday of October. November, April, and May.

Wichita Pediatric Society

President: Dr. Frank L. Menehan.
Secretary: Dr. R. C. Knappenberger.
Place: Homes of members. Time: 8:30 p. m., first Thursday, October through May.

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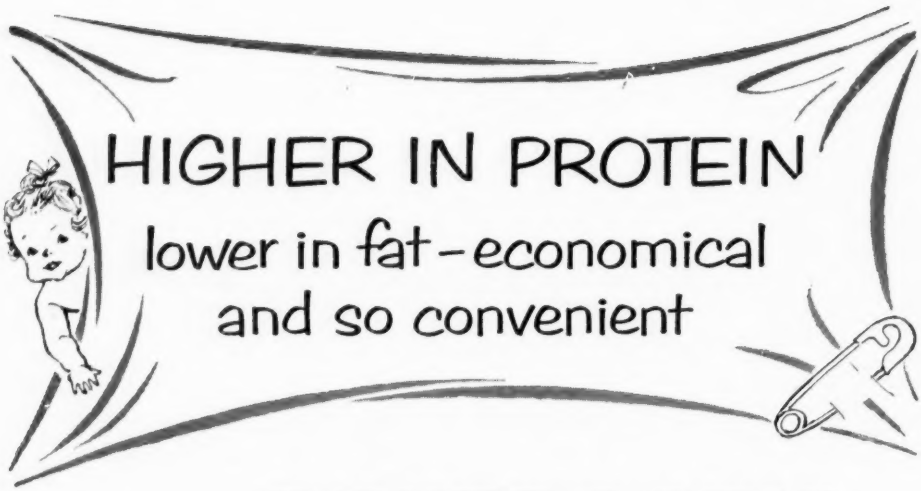

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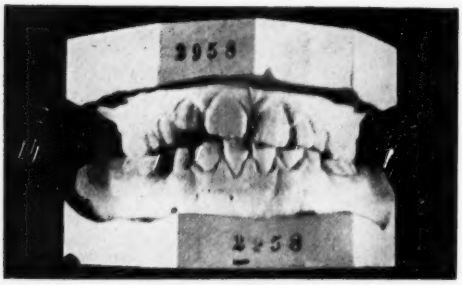

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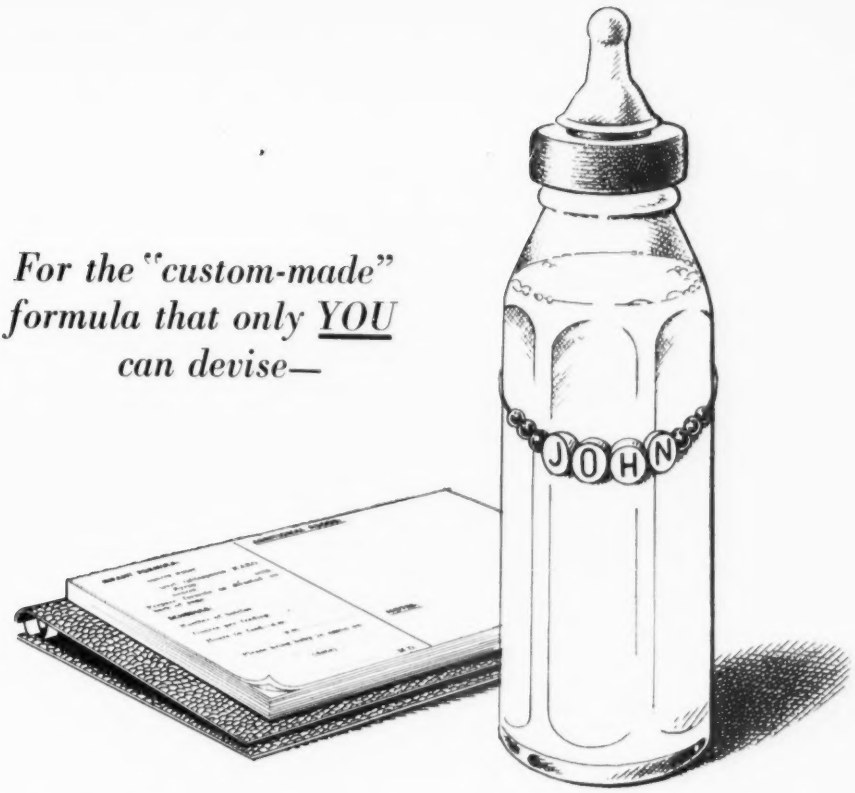

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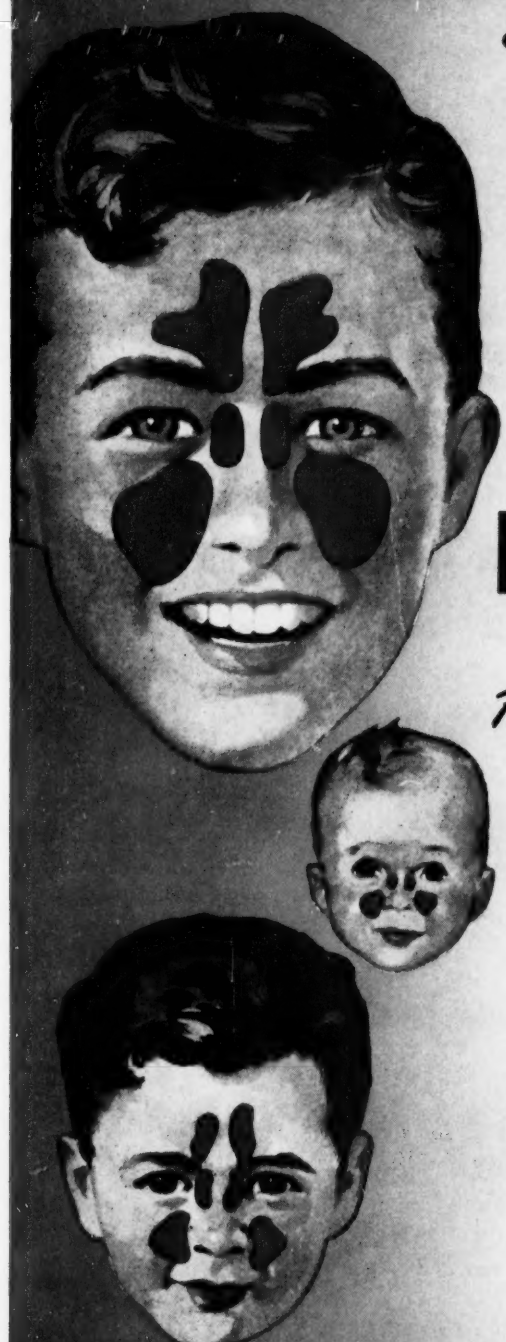
• IN INFANTS AND CHILDREN–



"In my hands the local treatment giving most satisfactory results is that originated by Proetz . . . displacement irrigation of the sinuses . . . Neo-Synephrine in normal saline . . . introduced into each nostril . . . These drainage and aeration operative procedures do no harm whatever to the physiology of the nose and will prevent more serious and destructive surgery in later life."

Burgess, T. S.: *Jour. Med. Assn. Georgia*, 37:44, Feb., 1948.

Neo-Synephrine®

HYDROCHLORIDE

BRAND OF PHENYLEPHRINE HYDROCHLORIDE

Prompt and Prolonged DECONGESTION

Through persistent decongestive action, Neo-Synephrine affords gratifying relief in colds and rhinitis as well as in sinusitis. It promptly checks mucosal engorgement and hypersecretion, permitting greater breathing comfort that lasts for hours.

Neo-Synephrine is notable for its relative freedom from sting, from compensatory congestion, from appreciable interference with ciliary action.

DOSAGE FORMS:

For nasal use

Neo-Synephrine HCl Solution **0.25%** (plain and aromatic) in 1 oz., 4 oz. and 16 oz. bottles
1% in 1 oz., 4 oz. and 16 oz. bottles
Water soluble jelly **0.5%** in ⅝ oz. tubes

For ophthalmic use

0.125% (⅛%), low surface tension, aqueous solution, isotonic with tears, in ½ oz. bottles

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